PLANT-LEVEL, SPATIAL, BIOECONOMIC MODELS OF PLANT DISEASE DIFFUSION AND CONTROL: GRAPEVINE LEAFROLL DISEASE

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SPATIAL, COMPUTATIONAL, BIOECONOMIC MODELS OF PLANT DISEASE DIFFUSION AND CONTROL: GRAPEVINE LEAFROLL DISEASE

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This dissertation addresses a family of agricultural and resource problems that emerge when biophysical and economic systems are endogenously linked over space and time. In particular, we study the spatial-dynamic bioeconomics of the grapevine leafroll disease (GLRD) which threatens the economic sustainability of the grape and wine industry. The first essay of the dissertation relies on a survey with vineyard managers in the Finger Lakes region of New York to assess the economic cost of the grapevine leafroll disease and identify optimal nonspatial disease control strategies under an array of bioeconomic parameters. The second essay employs cellular automata to model the disease spatial-dynamic diffusion for individual plants in a vineyard, evaluate nonspatial and spatial control strategies, and rank them based on vineyard expected net present values. Nonspatial strategies consist of removing and replacing symptomatic grapevines. In spatial strategies, symptomatic vines are removed and replaced, and their nonsymptomatic neighbors are virus-tested, then removed and replaced if the test is positive. We find that the nonspatial strategies currently recommended to the industry are not cost-effective under model baseline parameters. In contrast, we find that spatial strategies targeting immediate neighbors of symptomatic vines increase the vineyard expected net present value by 18-19%

relative to the strategy of no disease control. In the third essay, we model spatial-dynamic, negative externalities generated by a plant-level disease diffusion process between two ecologically connected but independently managed, heterogonous vineyards. One vineyard produces high-value wine grapes whereas the other produces low-value wine grapes. We embed the computational model in a bargaining game between neighboring managers. We find that, under noncooperative management, it is optimal for neither manager to control the disease. Under cooperative management, we find it optimal for the high-value manager to spatially control the disease and to pay the low-value manager to do similarly. The cooperative solution increases total payoffs by 20%. Using mean-preserving price expansions and contractions, we show that total payoff decreases with the magnitude of the price differential between the neighboring vineyards up to a point where cooperation becomes Pareto-optimal and the relationship between heterogeneity and total payoff becomes U-shaped.

BIOGRAPHICAL SKETCH

Shady S. Atallah holds an agricultural engineering degree from the Université Saint Esprit the Kaslik in Lebanon and a M.Sc. in Plant Science from the American University of Beirut. Prior to attending Cornell University for his Ph.D., Shady completed a M.Sc. in Agricultural and Resource Economics and a Postgraduate Certificate in Postharvest Biology and Technology, both from the University of California, Davis.

Shady has worked as an Agricultural Engineer and Project Officer for international non-governmental organizations in Lebanon and as a Middle East Agriculture Specialist for the International Programs of the University of California, Davis.

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To my parents, Salim and Yolla

To my fiancée, Catalina

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The first chapter of my dissertation is the result of field work, discussions, and co-authorship with Marc Fuchs (Department of Plant Pathology and Plant Microbe Biology) and Timothy Martinson (Department of Horticulture), of the New York State Agricultural Experiment Station. The formalization of the model in the second chapter has greatly benefited from consulting hours with James Booth (Department of Biological Statistics and Computational Biology and Cornell Statistical Consulting Unit).

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Many of the ideas in this dissertation were born from discussions with my then classmate, now fiancée Catalina Herrera-Almanza. Catalina and I have completed every step of the Ph.D. together starting with Math Camp in August 2009 and up to writing our dissertations now. This dissertation has greatly benefited from her intellectual input and her support.-

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INTRODUCTION

Grapevine leafroll disease (GLRD) presently threatens grape harvests in the United States and around the world. This viral disease is affecting the economic sustainability of the grape and wine industry by reducing yields and negatively affecting wine quality. GLRD is primarily introduced to vineyards through infected planting material. Once introduced, the disease can be transmitted by insect vectors within a vineyard block and between neighboring blocks or vineyards.

Vineyard managers are currently advised to avoid introducing GLRD into their vineyards by planting certified vines derived from virus-tested mother plants. Where GLRD is already present, disease management consists mainly of minimizing the source of inoculum by roguing symptomatic vines after harvest, especially the young ones and replacing them with virus-tested vines. With a few notable examples, little is known about the economic effects of GLRD. In fact, most GLRD research has focused on the pathogens with less work on disease ecology and disease management.

The case of GLRD diffusion and control in vineyards is a specific application of a family of agricultural and resource problems that are encountered when biophysical and economic systems are linked over space and time. These spatial-dynamic problems pose the familiar economic question of optimal effort allocation but focus on the spatial and temporal dimensions of resource management in the presence of spatial-dynamic externalities. In addition to providing results that are valuable to specific industries, this line of research addresses the increasing need of (and interest in) integrating the study of dynamic microeconomic and biophysical processes, over

both time and space, at the individual rather than the population level. Using GLRD as a case study, we offer a plant-level, spatial-dynamic, bioeconomic modeling approach that addresses these needs. From an industry perspective, this specific application is particularly important because it illustrates how the integration of spatial disease ecology in a plant-level, spatial-dynamic, bioeconomic model can inform the formulation of novel disease control strategies. These strategies are based on individual age-infection state, location, and plant neighborhood state, rather than visual symptoms alone. From a public policy perspective, this application improves our understanding of how agent heterogeneity affects strategic behavior and social welfare in the presence of negative, spatial-dynamic externalities.

This dissertation is comprised of three essays. In the first essay, we rely on a survey with Cabernet franc vineyard managers in the Finger Lakes region of New York to assess the economic cost of the grapevine leafroll disease under the various disease management scenarios encountered in the grape and wine industry. This essay identifies optimal disease control strategies based on initial disease incidence, extent of yield reduction, penalties imposed on fruit quality and vineyard age.

In the second essay, we examine whether a spatial-dynamic model that incorporates disease ecology parameters at the plant level could offer new insights on the optimal allocation of disease control effort over space and time. That is, insights that would not be possible to obtain from a nonspatial, model with biological parameters specified at the aggregate plant population level. In particular we examine whether optimal disease control is structured by age and is spatial. To do so, we employ cellular automata methods to model the disease spatial-dynamic diffusion at

the plant level in a single vineyard, evaluate nonspatial and spatial control strategies, and rank them based on vineyard expected net present values. We also ask whether and how results from such a model can guide research investments in GLRD disease ecology, plant pathology, entomology, and plant science.

In the third essay, we investigate how the optimal solution from the second essay is affected in the presence of spatial-dynamic, negative disease externalities. Here, we develop a plant-level model with distance- and density-dependent disease externalities between two ecologically connected but independently managed vineyards. The two neighboring wine grape growers are heterogeneous in the wine grape quality they produce, and consequently, in the prices they receive. We embed the computational model in a bargaining game between the grape growers and solve for the optimal disease management strategies in both vineyard blocks under central planner, noncooperative and cooperative disease management settings. We examine how heterogeneity affects strategic behavior in disease control and aggregate payoffs using prices for Cabernet Sauvignon in the Napa County wine production region in California.

CHAPTER 1
ECONOMIC IMPACT OF GRAPEVINE LEAFROLL DISEASE ON VITIS VINIFERA CV. CABERNET FRANC IN FINGER LAKES VINEYARDS OF NEW YORK

1.1 Introduction

Grapevine leafroll disease (GLRD) is one of the most widespread viral diseases in vineyards. It is reported in almost all grape and wine regions in the United States (Fuchs et al. 2009a, Golino et al. 2008, Martin et al. 2005) and worldwide (Charles et al. 2009, Freeborough and Burger 2006, Martelli and Boudon-Padieu 2006). GLRD causes significant yield losses (up to 30-68%), delays fruit ripening, reduces soluble solids and increases titratable acidity in fruit juice (Goheen and Cook, 1959, Martelli and Boudon-Padieu 2006, Martinson et al. 2008). Several phloem-limited filamentous viruses, designated as grapevine leafroll-associated viruses (GLRaVs), were isolated and characterized from leafroll-infected grapevines (Ghanem-Sabanadzovic et al. 2010, Martelli and Boudon-Padieu 2006). All GLRaVs are readily transmitted via vegetative propagation and grafting, and some of them (GLRaV-1, GLRaV-3, GLRaV-5 and GLRaV-9) are also vectored by several species of mealybugs (Hemiptera: Pseudococcidae) and soft scale insects (Hemiptera: Coccidae) (Martelli and Boudon-Padieu 2006, Tsai et al. 2010).

Vineyard managers adopt varied measures to manage GLRD (Martinson et al. 2008, Walker et al. 2004). Most of them tend to tolerate the disease without controlling it in spite of its evident detrimental impact on yield and fruit quality. Some managers, in contrast, replace infected vines with healthy ones (i.e. roguing), while a

few of them opt for replanting entire vineyards. Disease control decisions rely on a variety of factors but often do not take into account the impact on maturity and berry color at harvest. This may be explained by the fact that information about GLRD impact on profits is scarce. Therefore, vineyard managers may not make profit-maximizing decisions regarding GLRD control.

Little is known about the economic effects of GLRD, with a few notable examples. For instance, Walker et al. (2004) examined the impact of GLRaV-3 on gross margins in New Zealand vineyards using a model of virus spread under three infection scenarios (high, moderate and low) over six growing seasons. These authors estimated damages of approximately USD 21,200 per hectare by years 12, 15 and 17 for the high, moderate and low scenarios, respectively. Based on these results, the authors argued that replanting is justified in terms of increased profits by year 6, 8 and 11 for the same three scenarios, respectively. More recently, Nimmo-Bell (2006) employed a Net Present Value (NPV) approach to measure the economic costs of GLRaV-3 for *V. vinifera* cv. Sauvignon blanc and Merlot in New Zealand. The study compared the per-hectare NPV of infected and virus-free vineyard blocks under three scenarios of disease control: total vine removal in year 6, annual roguing of infected vines, and annual roguing of infected and neighboring vines. The authors concluded that early vine roguing is more cost-effective than total vineyard replacement in year 6. Vine roguing reduced the disease impact six-fold for Sauvignon blanc and seven-fold for Merlot when compared with the 'no intervention' scenario. In a study on the economic impact of GLRaV-3 on a *V. vinifera* Cabernet Sauvignon vineyard in the

Stellenbosch region of South Africa, Freeborough and Burger (2006) showed that roguing was the only viable alternative to increase profits.

In this study, we estimated the profitability impact of GLRD in grafted *V. vinifera* cv. Cabernet franc in Finger Lakes vineyards of New York. These estimates, in turn, were employed to recommend loss-minimizing management strategies for disease control. Specifically, the NPV approach was used to compare several GLRD control strategies, including roguing, replacing the entire vineyard and doing nothing. These management strategies were used to (1) quantify disease damage under several scenarios and, (2) identify optimal management strategies based on infection level, extent of yield reduction, penalties imposed on fruit quality and vineyard age.

1.2 Materials and Methods

1.2.1 Survey of vineyard managers. To construct economic analysis scenarios, a survey was conducted among ten vertically-integrated vineyard-winery operations in the Finger Lakes region of New York State during the period fall 2009 through spring 2010. The wineries selected had vineyards with a history of GLRD infection based on grafted *V. vinifera* cultivars, including cv. Cabernet franc, showing typical leafroll symptoms or infected with GLRaV -1, GLRaV-2 and/or GLRaV-3, as indicated by ELISA and/or RT-PCR (Fuchs et al. 2009b). Respondents provided information about perceived ranges of GLRD prevalence, magnitudes of yield reduction due to the disease, disease control measures adopted by vineyard managers, and penalties incurred due to poor fruit quality (see survey instrument in appendix). The survey

included data on all cultivars affected by GLRD. The analysis focused however on Cabernet franc for reasons outlined in the discussion section.

***1.2.2 Parameters used in construction of disease management scenarios*.** The survey responses were employed to specify the parameter ranges to be used in constructing the analysis scenarios as follows:

GLRD prevalence. Vineyard managers reported approximately 1, 5 and 40% levels of GLRD infection. These values, as well as other prevalence values retrieved from the literature, were considered to identify threshold levels that determine switching from one management option to another. Vineyard managers recognized that the occurrence of GLRD was mainly through infected vines at the time of planting.

Spread of GLRD by vectors. Managers did not recognize a pattern of virus spread via mealybug and soft scale insects, in spite of the documented presence of vectors species, including viruliferous individuals, in local vineyards (Fuchs et al. 2009b).The analysis employed a model of GLRaV-3 spread described in Charles et al. (2009) to predict levels of virus infection in the presence of vectors. In that model, a GLRD prevalence of 50% was predicted in years 6, 8 and 11 for the three vineyards studied with low, medium and high GLRD infection risk, respectively, and 90% in years 11, 12 and 15 (Charles et al. 2009). The data on GLRD spread over time in the medium infection risk case was used to construct this study's scenario of 'no GLRD control' (table 1.1, column N).

Table 1.1 GLRD prevalence and Cabernet franc yield over time

	Vines infected (%)					Yield (t/ha)		
Years	N[a]	T1[b]	T5	T40	T60	Healthy	50% reduction[c]	30% reduction[c]
0	0	-[d]	-	-	-	0	0	0
1	1	-	-	-	-	0	0	0
2	2	-	-	-	-	0	0	0
3	8	-	-	-	-	2.2	2.15	2.17
4	12	1	5	40	60	7.4	6.94	7.12
5	22	1	3	20	40	7.4	6.58	6.89
6	28	1	1	10	20	7.4	6.36	6.76
7	36	1	1	5	10	7.4	6.07	6.58
8	48	1	1	3	5	7.4	5.62	6.31
9	60	1	1	1	3	7.4	5.17	6.07
10	70	1	1	1	1	7.4	4.81	5.84
11	80	1	1	1	1	7.4	4.43	5.62
12	88	1	1	1	1	7.4	4.14	5.44
13	92	1	1	1	1	7.4	3.98	5.35
14	95	1	1	1	1	7.4	3.87	5.28
15-19	98	1	1	1	1	7.4	3.76	5.21
20-25	10	1	1	1	1	7.4	3.69	5.17

[a]N: GLRD infection levels derived from the model of Walker et al. (2004) for the no control scenario
[b]T1, T5, T40 and T60: roguing scenarios at 1, 5, 40 and 60% initial infection levels. GLRD prevalence under roguing was assumed to decrease following a stepwise pattern.
[c]Yield was calculated as %infected vines*yield of infected vines + %healthy vines*yield of healthy vines where yield reduction due to GLRD is assumed to be 50% and 30%
[d]It is assumed that GLRD is due to rootstock infection; therefore, 0-3 years old vines do not develop GLRD symptoms.

Methods of GLRD control. Vineyard managers practiced roguing (identifying, removing and replacing infected vines with vines derived from certified, virus-tested vines), replaced entire vineyards with virus-tested certified vines or did not respond to GLRD.

Yield reduction due to GLRD infection. For the most part, vineyard managers did not attempt to measure yield reduction due to GLRD. Therefore, the literature was reviewed and yield reductions of 30% and 50% were considered, given that 30-68%

losses are commonly reported (Goheen and Cook 1959, Martelli and Boudon-Padieu 2006, Martinson et al. 2008, Over de Linden and Chamberlain 1970).

Alteration of fruit quality due to GLRD infection. The survey quantified quality reduction due to increases in titratable acidity and reductions in sugar content of fruit juice at harvest. It also identified contractual mechanisms used by buyers to penalize poor quality grapes. Vineyard managers did not systematically measure the impact of GLRD on fruit juice chemistry. Instead, buyers inspected fruits and measured acidity and sugar levels. No vineyard manager reported rejection due to low quality grapes but one winery imposed a 10% price penalty when buying grapes that did not meet a sugar level requirement ranging from 15 to 21 Brix depending on the cultivar. For that reason, two additional scenarios were added (NN30 and NN50) in order to identify any effect of the penalty incentive on the vineyard managers' management decisions.

Integration of survey data. The answers provided by vineyard managers were used to identify parameter values to be considered in the analysis. These parameter values were combined to create scenarios under which the economic impact of GLRD was estimated. The scenarios are described below and summarized in table 1.2. Given that the study focused on Cabernet franc, data collected on GLRD incidence on other cultivars (section 2 of survey instrument in supplemental data) were not included in the analysis.

Table 1.2 Description of disease control scenarios

Scenarios	Initial infection level (%)	Yield reduction (%)			Quality penalty (%)		GLD introduction [a]		
		0	30	50	0	10	none	infected vines	vectors
Baseline (B)	0	x			x		x		
No disease control (N) [b]									
NN50	1			x	x			x	x
NN30	1		x		x			x	x
N50	1			x		x		x	x
N30	1		x			x		x	x
Planting virus-tested certified vines (C)	0	x			x		x		
Roguing scenarios (T1-T60)	1-60	x			x			x	
Vineyard replacement (R)	any	n/a[c]	n/a	n/a	n/a	n/a		x	x
No disease control, late vector-mediated infection (NLV)	0			x		x			x
Roguing, late vector-mediated infection (TLV)	0	x			x				x

[a] GLRD transmission within vineyards is not included here because it is assumed to be vector mediated in all scenarios
[b] NN50: No control, no penalty , 50% yield reduction; NN30: No control, no penalty , 30% yield reduction; N50: No control, 10% penalty, 50% yield reduction; and N30: No control, 10% penalty, 30% yield reduction
[c] Not applicable

1.2.3 Scenarios to assess GLRD impact. Various scenarios were constructed, reflecting the cash flow of one hectare of *V. vinifera* cv. Cabernet franc over 25 years (the typical lifespan of a vineyard in the Finger Lakes). These scenarios differ by the

biological and managerial parameters reported in the survey. Biological parameters include mode of disease transmission (through infected vines at time of planting for several levels of initial infection or, later on, through insect vectors) and various levels of yield impact (30 or 50% yield reduction). Management parameters, for their part, include disease control measures (no control, roguing at infection levels ranging from 1 to 60%, or entire vineyard replacement) and impact of the disease on price paid for the grapes due to quality losses (10% penalty or no penalty). Additional scenarios were considered in order to analyze how vineyard age might impact GLRD control decisions (ages 12, 16, and 20) and to assess the value of disease prevention by planting vines procured from certified, virus-tested stocks. The following scenarios were considered, based on the survey responses from vineyard managers:

Scenario 1: Baseline (B). The baseline scenario consists of a cash flow for one vineyard hectare over 25 years with no GLRD prevalence. The baseline scenario was employed as a benchmark to estimate the economic impact of GLRD under the scenarios described below. To this effect, the GLRD impact was computed as the difference between the baseline NPV and the NPV of each alternative scenario.

Scenario 2: No disease control (N). In this scenario GLRD is introduced in year one (either through insect vectors or at planting through infected vines at a level of 1%), spreads following the logistic model suggested by Charles et al. (2009), and the vineyard manager decides not to rogue or replace the vineyard. Disease spread is summarized in table 1.1 where column N lists the percentages of vines infected over time. This scenario was analyzed with a yield reduction of 30 and 50%, under either no penalty (NN 30 and NN 50) or a 10% penalty rate (N30 and N50) due to lower

quality grapes. This yielded four scenarios that were used to estimate the economic impact of GLRD when no control measures are implemented.

Scenario 3: GLRD prevention through establishing vineyards with planting material derived from certified, virus-tested stocks (C). This scenario simulates a situation where the vines used in a planting or replant site are derived from certified, virus-free stocks and cost 25% more than conventional vines, based on market prices. The NPV of this scenario is used to examine the benefits of a preventative approach to GLRD by procuring clean vines at the time of planting in situations where vines of poor sanitary status are the only source of infection.

Scenario 4: Roguing scenarios (T1-T60). These scenarios correspond to situations where GLRD is introduced at planting at different levels ranging from 1 to 60% (T1-60) via diseased rootstocks and/or scion. Infected vines start developing GLRD symptoms in year 4 and are subsequently rogued as they become symptomatic. Asymptomatic, infected vines are not identified nor removed and the disease can be re-introduced through insect vectors. Therefore, disease prevalence does not drop immediately but rather decreases in a stepwise pattern, as initially asymptomatic vines develop symptoms over time and are rogued. It is assumed that the disease is never eradicated and is controlled at 1% at best. The stepwise decrease in disease prevalence is formulated for roguing scenarios with varied initial infection levels (T1, T5, T40 and T60) (table 1.1). These parameters were used to identify the threshold infection level below which roguing is advisable and above which vineyard replacement is the appropriate response. In the roguing scenarios, it is assumed that there are no GLRD-led reductions in yield or quality.

Scenario 5: Vineyard replacement (R). In this scenario, the vineyard manager decides to replant the entire vineyard at the onset of symptoms in year 4. This scenario reflects actions of surveyed managers who are willing to invest in replanting in order to avoid the uncertainty of coping with GLRD and to minimize the probability of within-vineyard disease spread. The NPV of this scenario was used as a benchmark to identify infection level ranges that warrant vineyard replacement instead of roguing.

Scenario 6: Late vector-mediated GLRD infection (LV). In this scenario, GLRD is introduced through insect vectors in years 12, 16 or 20. It identifies a possible vineyard age beyond which no intervention would be recommended given a vineyard lifespan of 25 years.

Economic analysis. A NPV per hectare was calculated for each GLRD control scenario over the economic lifetime of vineyards (25 years). GLRD impacts were computed as the difference between the baseline NPV (i.e. no infection) and the NPV of the particular scenario considered. The NPV calculations are based on data (costs, revenues and financial assumptions) reported previously (White 2007), as described in table 1.3; on survey data (disease prevalence, impact on yield and price paid for the grapes) collected; and findings from the literature. Fixed costs were omitted from the analysis because they are identical for the different scenarios. Optimal control measures were identified as those with the highest NPV.

Table 1.3 Cabernet franc production, cost, revenue and financial assumptions

	Item	Value and unit
Production	Row spacing	2.7 m
	Vine spacing	1.8 m
	Planting density	1,994 vines/ha
	Vine replacement without GLRD	2%/year
Cost	Skilled labor wage	$16.6/hr
	Unskilled labor wage	$11.60/hr
	Gasoline	$0.76/L
	Diesel	$0.87/L
	Vines	$3.25/vine
Revenue	Price	$1,874/t
	Yield (years 4 and above)	7.4 t/ha
Financial	Discount rate	7.37%
	Project life cycle	25 years

Source: White (2007)

1.3 Results

The economic impact of GLRD over the lifetime of a Cabernet franc vineyard in the Finger Lakes ranges from $25,407 per hectare (for a 30% yield reduction and no quality penalty) to $41,000 per hectare (for a 50% yield reduction and a 10% penalty for poor fruit quality) if no control measures are implemented (table 1.4).

Value of planting vines derived from certified, virus-tested stocks. Results indicate that paying a price premium of 25% for clean plant material (i.e., 25% on top of the non-certified vine price of $3.25/vine, table 1.3) reduces GLRD-related losses to $1,829 per hectare. This loss is substantially smaller than those following roguing and vineyard replacement (table 1.5). It should be noted that this estimate does not take into account the possibility of a subsequent introduction of GLRD through vectors;

instead it focuses on the value of using certified vines to prevent the introduction of GLRD at planting.

Table 1.4 Net present value (NPV) of no GLRD control scenarios under different Cabernet franc yield reduction (30 and 50%) and quality penalty (0 and 10%) conditions

Scenarios	NPV[c] ($/ha)	GLRD economic impact[d] ($/ha)
No control, no penalty, 30% yield reduction (NN30)[a]	$7, 690	$25,407
No control, 10% penalty, 30% yield reduction (N30)	$6,786	$26,334
No control, no penalty, 50% yield reduction (NN50)	($7119)[c]	$40,241
No control, 10% penalty, 50% yield reduction (N50)	($7,900)	$41,019
Replacement[b]	$8,468	$24,651

[a] See description of scenarios in table 1.1
[b] Replacement: replacing vineyard at onset of symptoms in year 4
[c] Numbers in parenthesis represent losses
[d] Computed as the difference between the NPV of roguing and the baseline NPV

Roguing or vineyard replacement. The NPVs for roguing at various levels of initial infection and vineyard replacement suggest the existence of a threshold level of disease prevalence beyond which the optimal GLRD control is to replace the vineyard (Tables 5 and 6). Roguing yields higher NPVs than vineyard replacement for prevalence levels of 25% and below; and vineyard replacement yields the highest NPV for prevalence levels above 25%. The infection threshold is consistent with the survey responses. For example, a respondent that reported a 40% GLRD prevalence decided to replace the vineyard, whereas others dealing with infection levels of 1 and 5% practiced roguing. Under optimal GLRD control, the disease impact is reduced to

a range of $3,208 to $24,654 per hectare for scenarios of roguing at 1% GLRD prevalence and vineyard replacement, respectively (table 1.5).

Table 1.5 Economic impact of GLRD in a Cabernet franc vineyard under vine roguing, vineyard replacement, and planting virus-tested vines

Disease control scenarios[a]	NPV ($/ha)	GLRD economic impact[b] ($/ha)
Baseline scenario (B)	$33,122	$0
Establishing vineyard with certified, virus-tested vines (C)	$31,291	$1,829
Roguing scenarios (T1-60)		
1	$29,915	$3,207
5	$26,084	$7,038
10	$22,351	$10,771
20	$14,275	$18,847
25	$9,815	$23,307
26	$8,115	$25,007
30	$5,261	$27,861
40	$2,000	$35,121
50	($9,244)[c]	$42,366
60	($22,914)	$56,036
Vineyard replacement (R)	$8,468	$24,654

[a] See table 1.1 for scenario description
[b] The GLRD impact is computed as the difference between the NPV of roguing and the baseline NPV
[c] Numbers in parenthesis represent losses

Table 1.6 GLRD control decision matrix in a Cabernet franc vineyard based on yield reduction, GLRD prevalence and a quality penalty

Yield reduction level	Penalty level	
30% yield reduction	10% penalty	None
≤ 25% infection	rogue[a]	rogue
> 25% infection	replace vineyard	indifferent
Less than 30% yield reduction		
≤ 25% infection	rogue	rogue
> 25% infection	replace vineyard	do not control
50% yield reduction		
≤ 25% infection	rogue	rogue
> 25% infection	replace vineyard	replace vineyard

[a]Recommendations in decision matrix are based on results from Tables 4 and 5.

Late vector-mediated GLRD infection. Roguing has a positive impact on NPV even when infection occurs at a later stage. Roguing reduces losses by $9,271 and $11,733 per hectare in years 12 and 16, respectively (table 1.7). However, roguing increases losses in year 19 and after, suggesting the existence of a threshold vineyard age beyond which roguing is not optimal. This puts an upper bound on the age of the vineyard under which roguing remains economical; investing in planting new vines five years before the end of the lifecycle is not justified.

Table 1.7 NPV of scenarios depicting late vector-mediated transmission occurring in a Cabernet franc vineyard in years 12, 16 or 20 with (TLV)[a] and without roguing (NLV).

	NPV ($/ha)	Roguing impact[b] ($/ha)
Late vector-mediated infection scenarios		
Year 12, no roguing (NLV 12)	$23,502 [c]	
Year 12, roguing (TLV12)	$32,774	$9,272
Year 16, no roguing (NLV 16)	$18,286	
Year 16, roguing (TLV16)	$30,018	$11,733
Year 20, no roguing (NLV 20)	$31,118	
Year 20, roguing (TLV20)	$30,270	($848)[d]

[a]See table 1.1 for scenario description
[b]The impact of roguing is computed as the difference between the NPV of roguing and the NPV of 'no roguing'
[c]The NPVs are computed using infection levels from Table 2, column N
[d] Number in parenthesis represent losses

No control. Roguing is optimal for disease management with a 50% yield reduction because the 'no control' scenario yields negative NPVs (table 1.4, rows NN50 and N50). However, 'no control' was the most economically feasible response in certain instances. For example, for a 30% yield reduction and no penalty for poor fruit quality, the NPV (table 1.4, row NN30) equals the NPV of vineyard replacement (table 1.5). This result suggests that 'no control' is the optimal response when the level

of infection is above 25% (i.e. for the range where vineyard replacement is optimal), yield reduction is less than 30% and there is no quality penalty. The optimality of 'no control' in this case does not internalize the damages that could be caused by disease transmission to neighboring vineyards through insect vectors. In situations where there is evidence of such a transmission, replacing vineyard blocks with infection levels above 25% would be advisable to prevent secondary infections.

Changes in grape prices below and above the baseline price of $1,700/t did not lead to changes in the recommendation of roguing when GLRD prevalence is 25% and below and replacing the vineyard otherwise (table 1.8).

Table 1.8 Sensitivity analysis with respect to the price of Cabernet franc grapes: the recommendation of replacing the vineyard beyond 25% GLRD prevalence is unchanged

	NPV ($/ha)		
Disease control	p=$1,764/t	p=$1874/t	p=$1984.5/t
Roguing at 20% GLRD (T20)	$7,816	$14,275	$20,732
Roguing at 25% GLRD (T25)	$3,506	$9,815	$16,124
Roguing at 26% GLRD (T26)	$1,307	$8,115	$13,779
Roguing at 30% GLRD (T30)	($813)[a]	$5,261	$11,332
Replacing vineyard (R)	$2,439	$8,468	$14,498

[a]Number in parenthesis represent losses
p = price

1.4 Discussion

The economic impact of GLRD on grafted *V. vinifera* cv. Cabernet franc in New York (approximately $25,000-$40,000 per hectare) is consistent with losses reported by

Walker et al. (2004) (approximately $33,000-$50,000 per hectare by year 20 for three scenarios of infection risk) and Nimmo-Bell (2006) (approximately $47,000 per hectare) on *V. vinifera* cvs. Sauvignon blanc and Merlot in New Zealand. Sourcing clean, virus-tested vines reduces the economic impact of GLRD to a value that is below any of the NPVs of disease control (table 1.5). This finding suggests that vineyard managers should select virus-tested vines in order to maximize profits. Paying a premium of 25% on planting material derived from certified stocks is financially rewarding although it may not be attractive at first glance.

The estimated GLRD impact is particularly alarming for the Finger Lakes wine industry given the high prevalence of viruses associated with GLRD (Fuchs et al. 2009a, Martinson et al., 2008) and the documented presence of viruliferous insect vectors and their possible role in within-vineyard transmission (Fuchs 2008, Fuchs et al. 2009b). For example, 69% of the Cabernet franc vineyards surveyed in 2006 in the Finger Lakes was GLRD-affected (Martinson et al 2008). Applying this proportion to the Cabernet franc vineyards in the region (approximately 55 ha) results in 38 ha of infected vineyards. Based on NPVs (table 1.4), economic losses for that cultivar range from $1 to $1.5 million if the disease is not controlled. Evidence of high GLRD prevalence and presence of GLRaV vectors in the region is relatively recent. This might explain why some vineyard managers have underestimated GLRD-related losses. This study, along with the recent evidence of high GLRD prevalence, provides disease impact information that vineyard managers need to take into account to implement loss-minimizing disease control measures.

Although this study sheds light on the economic impact of GLRD, the results should be interpreted with caution. For example, disease spread patterns used in the study are from Charles et al. (2009) because no experimental data are available yet from the Finger Lakes. Given the low prevalence of mealybugs in the region (Fuchs et al. 2009a), the model could have overestimated the GLRD impact under the no control scenario. Future research should survey GLRD and vector prevalence over time to develop models of GLRD spread that can be used to estimate impact with more accuracy.

Earlier studies recommend controlling GLRD through roguing of symptomatic vines and their replacement with healthy ones (Freeborough and Burger 2006, Nimmo-Bell 2006, Walker et al. 2004). This study contributes to this literature by showing that roguing requires a large enough reduction in yield and/or enforcement of a price penalty on lower quality grapes to be economically justified. The 10% penalty rate reported in the survey may be too low and could underestimate the GLRD impact on wine quality. For example, Walker et al. (2004) assumed that grapes from infected vines lost 75% of their value. This is considerably higher than the penalty reported in the Finger Lakes (10% according to the survey) and might suggest that wineries underestimate the impact of GLRD on wine quality. Martinson et al. (2008) found that soluble solids were 2 Brix lower in grapes from GLRD-affected vines than from healthy vines. Those grapes also had higher juice pH and lower titratable acidity. Further sensory analysis studies are needed to link changes in Brix and acidity to changes in wine attributes and establish quality threshold levels for the of sugar and acidity contents. Then, using wine hedonic price models, a quality penalty could be

formulated in terms of ranges of sugar and acidity levels. Wineries would subsequently prevent a GLRD-related decrease in wine quality either by rejecting grapes that do not meet those thresholds or by imposing a quality penalty on those grapes and using them to produce bulk wines. This penalty can act as a price incentive for vineyard managers to control GLRD and prevent a loss in the market value of their grapes.

Over thirty wine grape cultivars are grown in the Finger Lakes. Although the analysis focuses on Cabernet franc, the results can be extended to other cultivars that are affected by GLRD in the region such as Cabernet Sauvignon, Chardonnay, Lemberger, Merlot, Pinot noir and Riesling (Fuchs et al. 2009a). Cabernet franc was particularly important to analyze for two reasons. First, GLRD symptoms are more visible in red than in white grape varieties (Martelli and Boudon-Padieu 2006) and roguing is therefore more easily implemented. Second, GLRD affects Cabernet franc more than other cultivars because it ripens later. This feature is crucial for cool-climate viticulture regions such as the Finger Lakes because the short growing season and early frost precludes delaying harvest to compensate for delayed ripening in GLRD-affected vines – which may be an option in areas with a longer growing season. In warmer climates and/or with early cultivars, it might be appropriate to replace the quality penalty component of the GLRD impact with a delayed harvest component and measure the economic losses associated with the delay, if any. GLRD impact on other cultivars would also be different due to differences in market prices; higher prices imply greater values of GLRD-related losses and vice-versa.

Among the available disease prevention and control methods, vector control was not included in the scenarios of this study since the efficiency of insecticides at reducing GLRD spread is still under study (Daane et al. 2008, Golino and Almeida 2008).

1.5 Conclusion

This research provides Finger Lakes vineyard managers with estimates of the economic impact of GLRD on the profitability of their businesses. It is estimated to range from $25,000 to $40,000 per hectare for scenarios of yield reduction and quality penalty over twenty-five years if left uncontrolled. The results suggest that, in order to minimize potential losses due to GLRD, managers ought to prevent infection by selecting certified, virus-tested vines for replant sites and by controlling the disease according to the decision matrix recommendations of this study. That is, disease control should be based on the values of infection level, yield reduction, price penalty incurred, and age of the vineyard. Future research should survey the prevalence of GLRD and its vectors over time to develop models that capture the disease dynamics in the Finger Lakes. Translating our results to other grape-growing regions with GLRD will require adjusting for differences in economic and epidemiological parameters that are unique to each region. However, independently of the region, we predict that roguing will remain the best control response up to a certain level of disease prevalence beyond which vineyard replacement will yield a higher net present value, and no disease control will be economically justified for certain parameter values.

CHAPTER 2

A PLANT-LEVEL, SPATIAL, BIOECONOMIC MODEL OF PLANT DISEASE DIFFUSION AND CONTROL: GRAPEVINE LEAFROLL DISEASE

Most GLRD research has focused on studying the pathogens with less emphasis on disease ecology and disease management (Almeida et al. 2013). This paper employs information available in the GLRD disease ecology literature to develop a computational, spatial bioeconomic model that can be used to identify profit-maximizing strategies for GLRD control. Using cellular automata, we model the disease at the plant level, in a spatial-dynamic way. In the simulations, the disease is introduced to an artificial vineyard through infected plant material at the time of planting. Subsequently, its diffusion follows a Markov process that is affected by each vine's location, virus detectability, age, own infection state, and infection states of its neighbors. We then use a vineyard manager's profit maximization objective function to evaluate the cost-effectiveness of disease control strategies formulated based on these vine-level characteristics. Our model contributes to the literature that employs nonspatial, compartmental models when modeling diseases by relaxing the simplifying assumptions that individuals are homogenous in their attributes and spatially perfectly-mixed. [1]

We examine the impact of alternative disease control strategies on distributions of bioeconomic outcomes and rank them based on the vineyard expected net present values (ENPVs). The results highlight the potential of vine-level, spatial

[1] The perfect-mixing assumption implies that any infective individual can transmit the infection to any healthy individual with equal probability (Brauer and Castillo-Chavez 2001).

strategies in reducing the economic cost of GLRD. In addition, our model can be modified to address spatial-dynamic disease diffusion and control issues in other perennial crops. We are not aware of previous work in agricultural and resource economics that formulates a spatial, plant-level, model of plant disease diffusion and control.

2.1 Literature Review

The unique characteristics of certain insect-transmitted plant diseases restrict the choice of approaches to model disease diffusion and control. The first characteristic of such diseases is that they are simultaneously driven by integrated dynamic and spatial forces, rather than by dynamic processes alone. When diseased plants are heterogeneously distributed in space and the physical environment includes spatial constraints on disease diffusion, such as a vineyard's spatial configuration, the optimality of disease control is affected not only by its intensity but also by its location.

Secondly, in insect-transmitted plant diseases, pesticide applications can be ineffective. This is particularly true in the case of GLRD where insect vectors can have a short infectivity retention period,[2] live in crevices and underneath the bark of the grapevine (Cabaleiro and Segura 2006, 2007; Daane et al. 2012), and can spread disease rapidly even if their population is kept at a low density (Charles et. al 2009; Tsai et al. 2008; Walton and Pringle 1999). Instead, insect-transmitted disease control relies mostly on reducing the source of infection by roguing (removing) infected

[2] The insect infectivity period is the time in which insect vectors retain the virus and remain infective (Tsai et al. 2008).

plants and replacing them with young, healthy ones (Chan and Jeger 1994). Thus, despite the attractive features of pest control models such as the ability to account for product quality in estimating pest control effectiveness (Babcock, Lichtenberg, and Zilberman 1992) or incorporating pest randomness in pesticide application decision rules (Saphores 2000), these models are not appropriate for vector-transmitted plant diseases such as GLRD.

Plant heterogeneity is the third characteristic of certain diseases. In the case of GLRD, individual vines that are infected but nonsymptomatic are heterogeneous in the time it takes for their virus population to be detectable by virus tests (Cabaleiro and Segura 2007; Constable et al. 2012). For some of these vines, the virus may not be detected and rogued before they transmit the disease to neighboring vines, causing disease control to lag behind disease diffusion and impeding eradication. Taken together, these three characteristics call for plant-level, spatial-dynamic models of disease diffusion and control.

2.1.1 Spatial Bioeconomic Models

Spatial-dynamic processes have only recently been studied by economists and the bioeconomic literature on agricultural diseases and invasive species control is mostly nonspatial (see review in Wilen 2007). Sanchirico and Wilen (1999, 2005) show that ignoring spatial processes can lead to suboptimal managerial decisions. Space can be incorporated in bioeconomic disease models by introducing barriers to disease diffusion (e.g., Brown, Lynch, and Zilberman 2002), specifying location-dependent, state-transition probabilities (e.g., Rich and Winter-Nelson 2007), or by using partial differential equations (e.g., Holmes et al. 1994). In such models, spatial heterogeneity

is exogenous and fixed over time (see review in Smith, Sanchirico and Wilen 2009). In some diseases including GLRD, however, spatial heterogeneity such as the health status of a plant's neighborhood can be endogenously determined by the diffusion process, affect disease diffusion and be affected by the implementation of control strategies. The challenge of incorporating such spatial feedbacks into state dynamics is a common thread in resource economics and not confined to disease dynamic models (Smith, Sanchirico and Wilen 2009). Moreover, spatial bioeconomic models often make restrictive assumptions such as linear growth and control to achieve tractability or to focus on steady state analyses in simple landscapes (see review in Epanchin-Niell and Wilen 2012). Relaxing such assumptions precludes analytical solutions and calls for numerical methods in most applications (Smith, Sanchirico and Wilen 2005; Wilen 2007).

2.1.2 Bioeconomic Models of Agricultural Diseases

Research on the economics of agricultural disease control has increasingly moved towards integrated epidemiological models that incorporate feedbacks between economic and disease diffusion components within the model (Beach et al. 2007; Fenichel and Horan 2007; Horan and Wolf 2005). These models typically aggregate individuals into disease-state (e.g. Horan et al. 2010) or age-state (e.g. Tahvonen 2009) compartments (they are thus called compartmental models), and employ differential or difference equations (DEs) to represent transitions between states. They assume that the population is spatially perfectly-mixed, and that the individuals are homogenous in their attributes within each compartment.

These assumptions are limiting in disease modeling, especially in the case of GLRD where (1) plants are heterogeneous in virus detectability, and (2) disease diffusion follows imperfect mixing processes and is shaped by vineyard spatial configuration and location of vines (Constable et al. 2012; Pietersen 2006). The homogeneity assumption of aggregate models is particularly restrictive because it precludes the formulation and testing of disease control strategies targeting individuals based on their heterogeneous, spatial-dynamic attributes. Also, the perfect-mixing assumption has been shown to underestimate the rate of spread in the early stages of a disease and to overestimate it in the later stages (Cane and McNamee 1982). These assumptions can be relaxed in DE models to represent distinct groups where individuals are heterogeneous by increasing the number of subpopulations or dividing the subpopulations into smaller stocks (e.g. Medlock and Galvani 2009). Depending on the level of heterogeneity desired, however, this process can lead to a combinatorial explosion in the number of state variables, equations, parameters, and data requirements (Teose et al. 2011). Moreover, in aggregate bioeconomic models of diseases, transmission rates are imposed on individuals exogenously depending on membership in a specific subpopulation. In reality, however, these rates are determined in a spatial-dynamic fashion as a result of the spatial-dynamic feedbacks between disease diffusion and disease control.

2.1.3 Cellular Automata Models

With dramatic decreases in computational costs, cellular automata and agent-based models have emerged as a preferred methodological framework to study complex systems (Miller and Page 2007) such as diseases. Cellular automata are dynamic

models that operate in discrete space and time on a uniform and regular lattice of cells. Each cell is in one of a finite number of states that get updated according to mathematical functions and algorithms that constitute state transition rules. At each time step, a cell computes its new state given its own old state and the old state of its neighborhood according to the transition rules (Tesfatsion 2006; Wolfram 1986). The spatial-dynamic structure is especially relevant when modeling processes that face physical constraints (Gilbert and Terna, 2000) such as boundaries and geometry as in the case of managed agricultural systems. In contrast with compartmental models, cellular automata and agent-based models do not aggregate individuals in compartments, thus allowing each individual to be heterogeneous in any finite number of attributes (Rahmandad and Sterman 2008). Although cellular automata models have been extensively employed to model spatial-dynamic processes (e.g. Sun et al. 2010; Yassemi, Dragićević, and Schmidt 2008), their use in the agricultural economics literature has been rare. The few examples include one application to the foot-and-mouth disease control (Rich, Winter-Nelson, and Brozovic 2005) and land use change studies (Balmann 1997; Kay-Blake et al. 2009; Marshall and Homans 2001; Roth et al. 2009).

We contribute to the disease control bioeconomic literature by employing cellular automata to offer a model that is inherently spatial and dynamic. We formally define the bioeconomic model, then build the computational model, verify its behavior, calibrate it and validate it using GLRD disease ecology literature field data. Using simulation experiments, we generate distributions of bioeconomic outcomes for the scenario of no disease, the strategy of no disease control and 18 alternative

nonspatial and spatial disease control strategies. We then conduct statistical analyses to rank the expected net present values generated in each experiment and find the optimal disease control strategy. Finally, we conduct sensitivity analyses to key bioeconomic model parameters. We synthesize our modeling process in table 2.1.

2.2 Bioeconomic Model

The spatial geometry of disease diffusion is represented by a two-dimensional grid G representing a vineyard plot. G is the set of *I* x *J* cells where *I* and *J* are the number of rows and columns, respectively. In our model, there are 5,720 cells $(i,j) \in G$, each holding one grapevine. Vineyard rows are oriented north to south with *I*=44 vines per grid row and *J*=130 vines per grid column resulting in a vineyard area of approximately 5.2 acres.[3]

[3] This configuration is considered representative of a typical vineyard in the Northeastern United States (Wolf 2008). The represented vineyard dimensions are 350' x 650'with an area of 227,500 ft^2 or 5.22 acres. Vine and column spacing are 5 and 8 feet, respectively.

Table 2.1 Overview of the Modeling Process

	Modeling Step	Tool
1.	*Formal model.*	
	Define bioeconomic model.	None.
2.	*Computational model.*	
2a.	*Model specification.*	Java, AnyLogic.
	- Specify cellular automata model by defining: o Space and time o States and state transitions - Define model parameters and variables.	
2b.	*Model verification.*	
	- Conduct simulation and collect simulated data. - Debug to ensure consistency in model behavior between computational model and formal model.	Java, AnyLogic.
2c.	*Model calibration*	
	Define optimization experiment that aims to find the optimal transmission parameter values using field data from the literature.	OptQuest, AnyLogic.
2d.	*Model validation*	
	Validate calibrated model by testing that the expected time to 50% disease prevalence (expected half-life) and expected time to 100% disease prevalence measures fall within intervals reported in the literature.	Java, AnyLogic.
3.	*Simulation experiments*	
	Define and conduct Monte Carlo experiments: scenarios of 'no disease', 'no disease control', 8 nonspatial and 10 spatial disease control strategies.	Java, AnyLogic.
4.	*Statistical analyses*	
	Conduct statistical tests on the differences between expected net present values.	Stata.
5.	*Sensitivity analyses*	
	Repeat steps 3 and 4 for each parameter considered in the sensitivity analysis.	

Each cell (i, j) has an age state and an infection state. Time t progresses in discrete monthly steps up to 600 months. $\boldsymbol{a}_{i,j,t}$ is a *600* × *1* vector holding a *1* for a vine's age in months and zeros for the other possible ages. A vine can be *Infective* or *Noninfective*. *Infective* (*I*) and *Noninfective* (*NI*) vines differ by whether or not they exhibit symptoms and have the ability to transmit the infection to their neighbors.

Noninfective vines, in turn, can be in the following infection states: *Healthy* (*H),* *Exposed-undetectable* (E_u)*,* and *Exposed-detectable* (E_d). Subdividing the *Noninfective* states allows us to separate healthy vines from those that have been exposed to the virus and have therefore lower grape yield and quality. The distinction between the states *Exposed-undetectable* (E_u)*,* and *Exposed-detectable* (E_d) is important to separate the vines whose virus populations have not reached detectable levels (*Exposed-undetectable*), from those with virus populations high enough to be detectable through a virus test (*Exposed-detectable*). *Infective* vines, for their part, can exhibit two states, namely *Infective-moderate* (I_m) or *Infective-high* (I_h). Separating the two states allows us to model the decrease in vine economic value as GLRD symptoms severity increases over time from the moderate to the high level. $\boldsymbol{s}_{i,j,t}$ is the infection state vector at time t of dimension 5×1. The vector holds a *1* for the state that describes a vine's infection state and zeros for the remaining four states. $\boldsymbol{w}_{i,j,t}$ is an age-infection composite state defined as the combination of a vine's age state $\boldsymbol{a}_{i,j,t}$ and its infection state $\boldsymbol{s}_{i,j,t}$.

A vine's infection and age states map into a third dynamic state variable, its economic value, or per-vine revenue $r(\boldsymbol{w}_{i,j,t})$. Per-vine revenue equals zero if the vine's age $\boldsymbol{a}_{i,j,t}$ is below τ_{max}. Beyond that age, $r(\boldsymbol{w}_{i,j,t})$ is function of the vine infection state. Grapes from GLRD-affected vines are subject to a penalty imposed on the price paid for grapes harvested from healthy vines. Furthermore, GLRD reduces grapevine yield by 30%, 50%, and 75% for vines in states *Exposed* (both *Exposed-undetectable* and *Exposed-detectable*), *Infective-moderate*, and *Infective-high*, respectively (table 2.2). A vine's revenue is known to a vineyard manager at time t.

Table 2.2 Vine Revenue

Age ($a_{i,j,t}$) and infection ($s_{i,j,t}$) states	Yield reduction (%)[a]	Quality penalty (%)[b]	Vine revenue ($/vine/month)[c] $r_{w_{i,j,t}}$
$a_{i,j,t} \leq 36$ months	n/a	n/a	0.00
$a_{i,j,t} \geq 48$ months and $s_{i,j,t} = H$	0	0	0.43
$a_{i,j,t} \geq 48$ months and $s_{i,j,t} = E_u$ or E_d	30	10	0.27
$a_{i,j,t} \geq 48$ months and $s_{i,j,t} = I_m$	50	10	0.19
$a_{i,j,t} \geq 48$ months and $s_{i,j,t} = I_h$	75	10	0.09

[a] Goheen and Cook (1959) and Martinson et al. (2008).
[b] Atallah et al. 2012.
[c] Vine revenue calculations are based on the Cabernet franc grape yield of 3.3 tons per acre, per year (White 2008), a planting density of 1096 vines per acre (Wolf 2008), and a grape price of $1,700/ton (White 2008).
n/a: not applicable (a vine is not productive below the age of 36 months).

Nevertheless, the per-vine revenue is random for periods beyond t as it depends on the vine's infection state $s_{i,j,t}$.

Given each vine's state $s_{i,j,t}$, and an infection state transition matrix $\boldsymbol{P}$, its expected infection state $\mathrm{E}(s_{i,j,t+1})$ at time $t+1$ is computed according to the following infection-state transition equation:

(1) $$\mathrm{E}(s_{i,j,t+1}) = \boldsymbol{P}^{\mathrm{T}}\, s_{i,j,t}$$

where E is the expectation operator and $\boldsymbol{P}^{\mathrm{T}}$ is the transpose of matrix $\boldsymbol{P}$. $\mathrm{E}(s_{i,j,t+1})$ is a *5* x *1* vector with a probability of staying in the current state, a probability of transitioning to the next state, and zeros elsewhere.

Disease diffusion is spatially constrained by the vineyard's horizontal (equation 2a) and vertical boundaries (equation 2b) as follows: [4]

(2a) $$(i-1) \in \{1, \ldots, I-1\}; (i+1) \in \{2, \ldots, I\};$$

(2b) $$(j-1) \in \{1, \ldots, J-1\}; (j+1) \in \{1, \ldots, J-1\}.$$

[4] These spatial constraints are formulated by defining the set of indices that vine (i,j)'s within-column (equation 2a) and across-column neighbors (equation 2b) can have. They ensure that the disease does not spread beyond the vines situated at the borders of the vineyard.

We now describe how the infection state transition probability matrix ***P*** governs disease diffusion. Vines in state *Healthy (H)* are susceptible to infection. They get exposed to the virus with a neighborhood-dependent probability *b*. At this point, they enter a latency period during which they are nonsymptomatic and noninfective. At first, the virus population in the vine is below levels that can be detected by virus tests and the vine is in state *Exposed-undetectable* (E_u). The virus population reaches detectable levels with probability *c* (i.e., the vine transitions to state *Exposed-detectable*, E_d). The transition to state *Infective-moderate* (I_m) happens with a probability d and marks the end of the latency period and the beginning of the infectivity period as well as the onset of visual symptoms. Symptoms, which consist of reddening and downward rolling of the leaves, are at moderate severity state first (I_m), and transition to a state of high severity later, or *Infective-high* (I_h), with a probability f. Mathematically, ***P*** can be expressed as follows:[5]

(3) $$\boldsymbol{P} = \begin{pmatrix} (1-b) & b & 0 & 0 & 0 \\ 0 & 1-c & c & 0 & 0 \\ 0 & 0 & (1-d) & d & 0 \\ 0 & 0 & 0 & (1-f) & f \\ 0 & 0 & 0 & 0 & 1 \end{pmatrix}$$

In equation (3), b is the *Healthy* to *Exposed-undetectable* transition probability conditional on previous own, and neighborhood infection states. It can be expressed as

[5] ***P*** reads from row (states *H*, E_u, E_d, I_m, and I_h at time *t*) to column (states *H*, E_u, E_d, I_m, and I_h at time *t+1*).

(4)

$$b = \Pr\left(s_{i,j,t+1} = E_u \,\middle|\, s_{i,j,t} = H\right) = \begin{cases} 0 \text{ if } s_{N_{i,j,t}} = (NI, NI, NI, NI) \\ 1 - e^{-\beta} \text{ if } s_{N_{i,j,t}} = (NI, NI, I, NI) \\ 1 - e^{-\beta} \text{ if } s_{N_{i,j,t}} = (NI, NI, NI, I) \\ 1 - e^{-2\beta} \text{ if } s_{N_{i,j,t}} = (NI, NI, I, I) \\ 1 - e^{-\alpha} \text{ if } s_{N_{i,j,t}} = (I, NI, NI, NI) \\ 1 - e^{-(\alpha+\beta)} \text{ if } s_{N_{i,j,t}} = (I, NI, I, NI) \\ 1 - e^{-(\alpha+\beta)} \text{ if } s_{N_{i,j,t}} = (I, NI, NI, I) \\ 1 - e^{-(\alpha+2\beta)} \text{ if } s_{N_{i,j,t}} = (I, NI, I, I) \\ 1 - e^{-\alpha} \text{ if } s_{N_{i,j,t}} = (NI, I, NI, NI) \\ 1 - e^{-(\alpha+\beta)} \text{ if } s_{N_{i,j,t}} = (NI, I, I, NI) \\ 1 - e^{-(\alpha+\beta)} \text{ if } s_{N_{i,j,t}} = (NI, I, NI, I) \\ 1 - e^{-(\alpha+2\beta)} \text{ if } s_{N_{i,j,t}} = (NI, I, I, I) \\ 1 - e^{-2\alpha} \text{ if } s_{N_{i,j,t}} = (I, I, NI, NI) \\ 1 - e^{-(2\alpha+\beta)} \text{ if } s_{N_{i,j,t}} = (I, I, I, NI) \\ 1 - e^{-(2\alpha+\beta)} \text{ if } s_{N_{i,j,t}} = (I, I, NI, I) \\ 1 - e^{-(2\alpha+2\beta)} \text{ if } s_{N_{i,j,t}} = (I, I, I, I) \end{cases}$$

In equation (4), $s_{N_{i,j,t}}$ is the infectivity state of a vine's von Neumann neighborhood.[6] For example, $s_{N_{i,j,t}} = (I, I, I, NI)$ is the state of a neighborhood composed of two *Infective* (*I*) neighbors in the same column (within-column neighbors), one *Infective* neighbor in the adjacent column (across-column neighbor) and one *Noninfective* (*NI*) neighbor in the other adjacent column (across-column neighbor). Given that each of the four neighbors can be in one of two infectivity states (*I* or *NI*), $s_{N_{i,j,t}}$ can be in one of the 2^4 states listed in equation (4). Parameters α and β are the within-column and across-column transmission rates with $\alpha > \beta > 0$,

[6] This type of neighborhood represents the most common vertical trellis system where a vine is in contact with its four neighbors in the cardinal directions. In contrast, a horizontal trellis system favors contact with up to eight neighbors (Cabaleiro and Segura 2006) and could be represented by a Moore neighborhood.

suggesting that *Infective* vines transmit the disease to their neighbors within the grid column at a higher rate than they transmit it to their neighbors situated in the adjacent grid column. We choose this neighborhood-based infection state transition to reflect patterns of GLRD diffusion observed in spatial analyses where the disease is shown to spread preferentially along grid columns (Habili et al. 1995; Le Maguet et al. 2013). We assume that within- and across-column infections occur independently with rate parameters α and β. That is, the time a vine stays in the *Healthy* state before transitioning to the *Exposed-undetectable* state, is a random variable, exponentially distributed, with rate α for within-column state transitions and rate β for across-column state transitions. In each time step, a random variable u_t determines whether the *Healthy* to *Exposed-undetectable* state transition happens or not. A *Healthy* vine that has one within-column *Infective* neighbor (e.g., $s_{N_{i,j,t}} = I, NI, NI, NI$) receives the infection at time *t+1* if $u_t < 1 - e^{-\alpha}$, where u_t is a random draw from $U \sim (0, 1)$. Conversely, the disease is not transmitted if $u_t \geq 1 - e^{-\alpha}$. Similarly, a *Healthy* vine that has one across-column *Infective* neighbor (e.g., $s_{N_{i,j,t}} = NI, NI, I, NI$) receives the infection at time *t+1* if $u_t < 1 - e^{-\beta}$ and is not transmitted if $u_t \geq 1 - e^{-\beta}$. When two or more transmission types are realized- for example, when a vine has one *Infective* within-column neighbor and one *Infective* across-column neighbor- the disease transmission is determined by the shortest of the waiting times (Cox 1959).[7]

The probability of transition from *Exposed-undetectable (E_u) to Exposed-detectable (E_d)* is given by c as follows:

[7] We compare the random variable with the transition probability in each time step because the state of a vine's neighborhood is changing over time, thus changing the probability that a vine receives the infection in each time interval.

(5) $$c = \Pr(X < x) = \begin{cases} 0 & \text{if } x < m_1 \\ \frac{(x-m_1)^2}{(m_2-m_1)(m_3-m_1)} & \text{if } m_1 \leq x \leq m_3 \\ \left(1 - \frac{(m_2-x)^2}{(m_2-m_1)(m_2-m_3)}\right) & \text{if } m_3 \leq x < m_2 \\ 1 & \text{if } x > m_2 \end{cases}$$

This transition happens after a vine has spent a period X in state *Exposed-undetectable*. Cabaleiro and Segura (2007) and Constable et al. (2012) report minimum (m_1), maximum (m_2) and most common (m_3) values for this period. With no further knowledge on the distribution of this period, we assume it is drawn from a triangular distribution with parameters $m_1, m_2,$ and m_3. The probability that the transition happens in less than *x* time steps, or $\Pr(X < x)$, can then be written as a function of these parameters (Kotz and van Dorp 2004).

The *Exposed-detectable* to *Infective* state transition probability is given by d as follows:

(6) $$d = \Pr\left(s_{i,j,t+1} = I_m \,|\, s_{i,j,t} = E_d\right) = \begin{cases} 1 - e^{-1/L_y} & \text{if } a_{i,j,t} = Young \\ 1 - e^{-1/L_m} & \text{if } a_{i,j,t} = Mature \\ 1 - e^{-1/L_o} & \text{if } a_{i,j,t} = Old \end{cases}$$

This probability depends on a vine's age category. Younger vines have shorter latency periods (Pietersen 2006), i.e. $L_y < L_m < L_o$, where subscripts y, m and o denote young (0-5 years), mature (5-20 years) and old (>20 years) vines, respectively. The waiting time after a vine enters state *Exposed-detectable* (E_d) and before it transitions to state *Infective moderate* (I_m) is a random variable, exponentially distributed with fixed rate parameter $1/L_y$ for young vines, $1/L_m$ for mature vines, and $1/L_o$ for old vines.

Finally, once a vine is in state *Infective moderate* (I_m), symptom severity increases over time and reaches a high level after a period *Inf*. That is, the waiting time until a vine transitions from *Infective-moderate* (I_m) to *Infective-high* (I_h) is a random

variable, exponentially distributed with fixed rate parameter $1/Inf$. Thus, the probability that a vine transitions from I_m to I_h

is $f = \Pr(s_{i,j,t+1} = I_h \mid s_{i,j,t} = I_m) = 1 - e^{-1/Inf}$.[8] Symbols, definitions, values, and references for the model parameters are presented in table 2.3.

The objective of a risk-neutral vineyard manager is to maximize the vineyard ENPV by choosing an optimal disease control strategy from a discrete set of spatial and nonspatial strategies, $\mathcal{W}$. Strategies are based on vines' age-infection states. Each strategy translates into two binary decisions for each vine in cell (i, j) in each time step. The first vine-level decision a manager faces is whether to rogue a vine and replace it with a new, virus-free vine [9] ($u_{w_{i,j,t}} = 1$ if roguing and replanting takes place, 0 otherwise). The other decision is whether to test for the virus ($v_{w_{i,j,t}} = 1$ if virus testing takes place, 0 otherwise).

The optimal strategy $\mathcal{W}^*$ is the sequence of vine-level control variables $\{u_{w_{i,j,t}}, v_{w_{i,j,t}}\}$ that allocates disease control effort over space and time so as to yield the maximum ENPV improvement over the strategy of no disease control. Letting $\mathbf{E}$ be the expectation operator over the random vine-level revenue $r(w_{i,j,t})$, ρ^t the discount factor [10] at time t (in months) where $t \in \{0, 1, 2, \ldots, 600\}$, the objective of a vineyard manager is to maximize the expected net present value (ENPV) as follows:

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[8] Note that, we do not need to compare a random variable with the probabilities at each time interval for the E_u-to- E_d, E_d-to- I_m, and I_m-to-I_h transitions because the rates and waiting times in these transitions do not depend on the neighborhood state and are therefore fixed.

[9] We assume that the vineyard manager follows industry recommendations and uses virus-tested vines when replacing an infected vine. We also assume that virus-tested vines are virus-free.

[10] $\rho^t = \frac{1}{(1+r)^t}$, where r is the discount rate.

Table 2.3 Model Parameters

Parameter	Description	Value	Unit	Sources
α	Within-column H to E_u transition rate.	4.2[a]	month $^{-1}$	Calibration experiment based on Charles et al. (2009).
β	Across-column H to E_u transition rate.	0.014[a]	month $^{-1}$	
L_y	Latency period for young vines.	24	months	Age-specific latency periods based on Jooste, Pietersen, and Burger (2011).
L_m	Latency period for mature vines.	48	months	
L_o	Latency period for old vines.	72	months	
m_1	Minimum of undetectability period.	4	months	Cabaleiro and Segura (2007); Constable et al. (2012).
m_2	Maximum of undetectability period.	18	months	
m_3	Mode of virus undetectability period.	12	months	
Inf	Period spent in state I_m before a vine transitions to state I_h.	36	months	M. Fuchs, personal communication, April 9, 2012.
τ_{max}	Period from planting until productivity.	36	months	White (2008).
ρ	Discount factor.	0.9959	month $^{-1}$	Assumed. Equivalent to an annual discount rate of 5%.
$c_{u_{i,j}}$	Unit cost of vine roguing (removal) and replacement.	7.25	\$/vine	Based on White (2008) and Atallah et al. (2012);
$c_{v_{i,j}}$	Unit cost of vine virus testing.	2.61	\$/vine	AC Diagnostics (2012) for the material cost based on 1,000 samples; Luminex (2010) for the labor time.

[a] Transition rates are constant for a particular location over the 50 year period of study. This excludes for instance situations where new insect vector species are introduced and contribute to an increase in transmission rates.

(7) $\max_{W} \mathrm{E} \sum_{t\in T} \rho^{t} \sum_{(i,j)\in G} \left\{ \left(1 - \sum_{\tau=0}^{\tau_{max}} u_{w_{i,j,t-\tau}}\right) r(w_{i,j,t}) - \left(u_{w_{i,j,t}} c_{u_{i,j}} + v_{w_{i,j,t}} c_{v_{i,j}}\right) \right\}$

subject to the infection state transition equation (equation 1) and the spatial constraints to disease diffusion (equations 2a and 2b). Note that the objective function not only takes into account the total amount of control realized under each strategy but also the timing, intensity and location of that control. The first expression in the curly brackets of equation (7) represents the revenue of a vine in location (i,j) and in age-infection state $\boldsymbol{w}_{i,j,t}$ at time *t*. If a manager has decided to rogue and replant vine (i,j) in the last τ_{max} periods, then $u_{w_{i,j,t-\tau}}$ is equal to 1 and the revenue is pre-multiplied by zero for a period of τ_{max} until the replant bears fruit, where $\tau \in \{1,2,\ldots,\tau_{max}\}$, and $\sum_{\tau=0}^{\tau_{max}} u_{w_{i,j,t-\tau}} = \{0,1\}$.[11] The second expression in the curly brackets represents the cost of roguing-and-replanting ($c_{u_{i,j}}$), and the cost of testing ($c_{v_{i,j}}$), pre-multiplied by their corresponding binary decision variables.

2.2.1 Model Initialization

At the beginning of a simulation, 2% of the grapevines (including those situated at the border of the vineyard) are randomly chosen from a uniform spatial distribution *U* (0, 5720) to transition from the *Healthy* to the *Exposed* state. This reflects findings in GLRD studies indicating that primary infection sources are randomly spatially distributed (Cabaleiro et al. 2008), and that initial disease prevalence is typically between 1% and 5% (Atallah et al. 2012). Thereafter, GLRD spreads to uninfected vines.

[11] This condition says that roguing and replanting in cell (i,j) cannot occur more than once in τ_{max} periods. It implies that a replant is never rogued before it bears fruit.

2.2.2 Model calibration and parameterization

In order to select the disease transmission parameter values (α and β in table 2.3), we first define a calibration objective function that minimizes the difference between the total number of infected vines over time obtained from our computational model (under no disease control), and the total number of infected vines over time from temporal disease progress data in Charles et al. (2009). Next, we use an optimization engine (OptQuest ^{TM}) that varies the values of α and β in each of the Monte Carlo simulations according to an algorithm combining Tabu search,[12] scatter search, integer programming and neural networks, to find the optimal parameter values (Step 2c. in table 2.1). We finally validate that the expected time until 50% disease prevalence (vineyard half-life) and expected time to 100% disease prevalence measures from the calibrated model fall within ranges of temporal disease diffusion curves reported in the GLRD literature (Cabaleiro and Segura 2006; Cabaleiro et al. 2008) (Step 2d., table 2.1). For the other parameters, we choose values reported in the literature after consultation with GLRD experts (table 2.3).

We choose a monthly time step because it gives the disease diffusion model a temporal resolution that is consistent with the magnitude of the disease diffusion parameters. With no information on diffusion seasonality, we do not model seasons and assume for simplicity that disease diffusion and control can take place in any month.[13]

[12] Tabu search is a metaheuristic procedure for solving optimization problems, designed to guide other methods to avoid the trap of local optimality.

[13] Note that, although simplifying, this assumption is consistent with the fact that, in reality, both disease diffusion and disease control take place during the same months (the growing season).

2.3 Experimental Design

We design and implement Monte Carlo experiments to evaluate nonspatial and spatial disease control strategies by comparing their bioeconomic outcomes to those resulting from a strategy of no disease control. Each experiment consists of a set of 1,000 simulation runs, over 600 months, on a vineyard of 5,720 grapevines. Experiments differ in the disease control strategy they employ. Outcome realizations for a run within an experiment differ due to random spatial initialization and random spatial disease diffusion. Data collected over simulation runs are the expected values of the bioeconomic outcomes under each strategy (Step 3, table 2.1). Finally, we conduct statistical tests to rank the disease control strategies and find the optimal strategy (Step 4, table 2.1). The model is written in Java and simulated using the software AnyLogic™ (XJ Technologies). Below, we describe the disease control strategies and the outcomes measured.

2.3.1 Disease control strategies

The discrete set of disease control strategies, w, includes 18 spatial and nonspatial strategies, in addition to the strategy of no disease control. In the subset of *nonspatial strategies*, the vineyard manager decides whether to rogue and replace symptomatic vines based on their symptoms (*Infective-moderate; Infective-high*) with or without considering their age (*Young*: 0-5; *Mature*: 6-19; *Old*: 20 and above). There are eight nonspatial strategies (Strategy 1 to Strategy 8 in table 2.4). [14] In the subset of *spatial strategies*, the vineyard manager decides whether to rogue and replant vines as soon as

[14] We exclude the strategy of roguing and replacing *Infective-high* and *Young* (I_hY) because this age-infection combination cannot be reached; it takes a vine more than 5 years to transition to the *Infective-high* state.

they develop symptoms (*Infective-moderate*), test their neighbors and rogue-and-replace them if they test positive. Figure1 shows the four types of neighborhoods considered in disease control (panels a, b, c, and d). If a vine tests positive, it is removed in the same period. There are 10 spatial strategies (Strategy 9 to Strategy 18 in table 2.4). The scenario of no disease provides a baseline to compute the expected disease economic cost under each candidate strategy.

2.3.2 Bioeconomic outcomes measured and ranking of control strategies

In order to find the optimal disease control, we employ the objective function (equation 7) to rank the vineyard net present values under the alternative strategies. In addition, we collect simulated data on the expected total disease control costs, the expected total number of grapevines rogued and replaced, the expected average vineyard age, and the expected half-life. The latter is defined as the expected time period to reach 50% disease prevalence. It is a measure of the ability of a control strategy to slow down disease diffusion.

2.4 Results and Discussion

Overall, we find that spatial strategies are superior to nonspatial strategies. In fact, none of the nonspatial strategies improve the expected net present value over the strategy of no disease control, under the base model parameter values. We also find that age-structured disease control improves a vineyard ENPV compared to nonage-structured control. However, such improvements are not present with spatial strategies. Among nonspatial strategies, targeting young, moderately infected vines (Strategy I_mY) yields the highest vineyard ENPV. The spatial strategy of targeting

symptomatic vines and their four immediate neighbors (Strategy I_mNSEW) is optimal, maximizing the vineyard ENPV.

Figure 1. Types of grapevine neighborhood

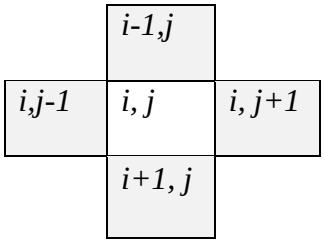


Fig. 1a Vines targeted under Strategy I_mNSEW (vine (i, j)'s von-Neumann neighborhood)

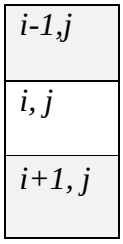


Fig. 1b Vines targeted under Strategy I_mNS

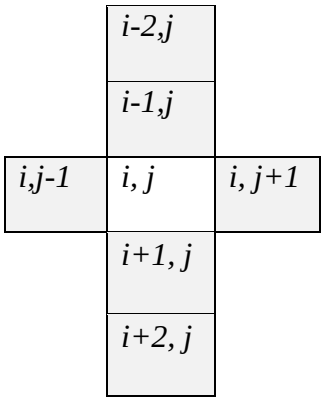


Fig. 1c Vines targeted under strategy I_mNS2EW

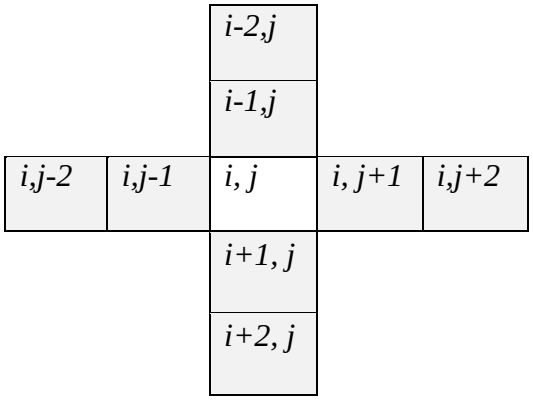


Fig. 1d Vines targeted under strategy $I_mNS2EW2$

Table 2.4 Disease Control Strategies: definitions and acronyms

	Disease Control Strategies	Acronyms
Nonspatial strategies[a].		
1	Roguing and replacing all vines that are *Infective.*	I
2	Roguing and replacing all vines that are *Infective-moderate.*	I_m
3	Roguing and replacing all vines that are *Infective-high.*	I_h
4	Roguing and replacing vines that are *Infective-moderate* and *Young.*	I_mY
5	Roguing and replacing vines that are *Infective-moderate* and *Mature.*	I_mM
6	Roguing and replacing vines that are *Infective-moderate* and *Old.*	I_mO
7	Roguing and replacing vines that are *Infective-high* and *Mature.*	I_hM
8	Roguing and replacing vines that are *Infective-high* and *Old.*	I_hO
Spatial strategies[b].		
9	Roguing and replacing *Infective-moderate* vines in addition to testing their two within-column neighbors then roguing and replacing those that test positive.	I_mNS
10	Roguing and replacing *Infective-moderate* vines in addition to testing their two across-column neighbors and two-within column neighbors then roguing and replacing those that test positive.	I_mNSEW
11	Roguing and replacing *Infective-moderate* vines in addition to testing their four within-column neighbors and two across-column neighbors then roguing and replacing those that test positive.	I_mNS2EW
12	Roguing and replacing *Infective-moderate* vines in addition to testing their four within-column and four within-row neighbors then roguing and replacing those that test positive.	$I_mNS2EW2$
13	Roguing and replacing *Young, Infective-moderate* vines in addition to testing their two within-column neighbors then roguing and replacing those that test positive.	I_mY-*NS*
14	Roguing and replacing *Mature, Infective-moderate* vines in addition to testing their two within-column neighbors then roguing and replacing those that test positive.	I_mM-*NS*
15	Roguing and replacing *Old, Infective-moderate* vines in addition to testing their two within-column neighbors then roguing and replacing those that test positive.	I_mO-*NS*
16	Roguing and replacing *Young, Infective-moderate* vines in addition to testing their two across-column neighbors and two-within column neighbors then roguing and replacing those that test positive.	I_mY-*NSEW*
17	Roguing and replacing *Mature, Infective-moderate* vines in addition to testing their two across-column neighbors and two-within column neighbors then roguing and testing those that test positive.	I_mM-*NSEW*
18	Roguing and replacing *Old, Infective-moderate* vines in addition to testing their two across-column neighbors and two-within column neighbors then roguing and replacing those that test positive.	I_mO-*NSEW*

[a] Note that strategies 4 to 8 are the age-structured counterparts of strategies 2 and 3.
[b] Note that strategies 13 to 18 are the age-structured counterparts of strategies 9 and 10.

2.4.1 Nonspatial strategies

Our simulations indicate that the vineyard's ENPVs over a 50-year period are higher when nonspatial disease control strategies are structured by age (I_mY, I_mM, I_mO, I_hM, I_hO) compared to the strategies that are not (I_m, I_h, I) (table 2.5). Structuring strategies by vine age increases revenues by reducing the number of unproductive replants and disease control costs compared to nonage-structured strategies. The strategy of targeting young, moderately infected vines (Strategy I_mY) yields the highest expected net present value. This result is significant because a vineyard manager might be inclined to wait until a productive vine is more severely infected and older before roguing and replacing it. Doing so, however, causes roguing and replanting to occur too late and less frequently thus reducing the vineyard ENPV.

Under the base model parameter values, none of the nonspatial strategies yield a positive ENPV improvement over the strategy of no disease control. This finding suggests that current industry recommendations for roguing and replanting all symptomatic vines might not be ENPV-improving if the within-column disease transmission is high enough. Our result depends critically on the baseline value of the within-column transmission parameter α. We decrease this value to find a threshold that renders the best nonspatial Strategy I_mY (targeting young, moderately infected vines) ENPV-improving. We find that, when α equals 1.169, Strategy I_mY yields an ENPV that is 3% higher than of the ENPV of no disease control. This improvement is statistically significant at the 1% level.

Table 2.5 Disease Control Strategies: Expected Net Present Value Improvements

	Disease Control Strategies	Acronyms	Expected Net Present Values [a] Value	Disease Cost [b]	Improvement over 'no disease control' [c]	
			$1,000	*$1,000*	*$1,000*	*%*
	No disease		463 (0)[d]	n/a	n/a	n/a
	No disease control		316 (4)	147	n/a	n/a
Nonspatial strategies						
1	All *Infective*	I	-147 (16)	610	-463***	-147
2	*Infective-moderate*	I_m	-146 (16)	609	-462***	-146
3	*Infective-high*	I_h	284 (4)	179	-32***	-10
4	*Infective-moderate* and *Young*	I_mY	314 (3)	148	-1***	0
5	*Infective-moderate* and *Mature*	I_mM	309 (4)	154	-7***	-2
6	*Infective-moderate* and *Old*	I_mO	308 (4)	155	-8***	-2
7	*Infective-high* and *Mature*	I_hM	295 (4)	168	-21***	-7
8	*Infective-high* and *Old*	I_hO	310 (4)	153	-6***	-2
Spatial strategies						
9	Two within-column neighbors of *Infective-moderate* vines.	I_mNS	374 (6)	89	58***	18
10	Two within- and two across-column neighbors of *Infective-moderate* vines.	I_mNSEW	374 (5)	88	59***	19
11	Four within- and two across-column neighbors of *Infective-moderate* vines.	I_mNS2EW	309 (4)	154	-7***	-2
12	Four within- and four across-column neighbors of *Infective-moderate* vines.	$I_mNS2EW2$	295 (5)	168	-21***	-7
13	Two within-column neighbors of *Young, Infective-moderate* vines.	I_mY-NS	215 (5)	248	-101***	-32
14	Two within-column neighbors of *Mature, Infective-moderate* vines.	I_mM-NS	316 (5)	147	0.1***	0
15	Two within-column neighbors of *Old, Infective-moderate* vines.	I_mO-NS	303 (4)	159	-13***	-4
16	Two within- and two across-column neighbors of *Young, Infective-moderate* vines.	I_mY-$NSEW$	-59 (14)	521	-374***	-119
17	Two within- and two across-column neighbors of *Mature, Infective-moderate* vines.	I_mM-$NSEW$	317 (4)	146	1***	0
18	Two within- and two across-column neighbors of *Old, Infective-moderate* vines.	I_mO-$NSEW$	302 (4)	161	-14***	-4

n/a is not applicable.
[a] Expectations are obtained from 1,000 simulations for the 5-acre vineyard.
[b] Expected Disease Cost = ENPV ('No disease')- ENPV (Strategy).
[c] ENPV improvement = ENPV (Strategy) - ENPV ('No disease control').
[d] Standard deviations in parentheses.
*** Difference is significant at the 1% level.

2.4.2 Spatial Strategies

Among the nonage-structured spatial strategies (Strategies 9 through 12), the one that targets symptomatic vines and their four immediate neighbors (Strategy I_mNSEW) yields the highest ENPV improvement over the strategy of no disease control ($59,000 or 19%, table 2.5). This improvement is statistically significant at the 1% level. This strategy reduces the vineyard-level disease cost to $88,000 over 50 years, which is smaller than the cost of $147,000 when the disease is not controlled and the cost of $148,000 when the best nonspatial strategy is employed (Strategy I_mY). The second-best strategy is I_mNS, which targets symptomatic vines and their two immediate neighbors. This strategy yields an ENPV improvement of $58,000 and reduces the disease cost to $89,000. This improvement is statistically significant at the 1% level. These results underscore the benefits of spatial disease control strategies in comparison with nonspatial strategies. Spatial strategies I_mNS and I_mNSEW increase the vineyard ENPV through early detection and control of nonsymptomatic grapevines situated in the neighborhood of a symptomatic grapevine. Consequently, the *Infected-high* state is never reached and the highest yield reduction (75%, table 2.2, I_h) is never realized. At the terminal period, disease prevalence is 13% and 4% under Strategy I_mNS and Strategy I_mNSEW, respectively.

The disease economic cost under the optimal Strategy I_mNSEW is approximately $25,000 per hectare by year 25, and contrasts with previous GLRD estimates of approximately $8,000 per hectare over 20 years (Nimmo-Bell, 2006) and $7,000 per hectare over 25 years (Atallah et al. 2012). These studies assume that a strategy consisting of roguing and replanting all symptomatic vines is capable of

reducing the disease to a prevalence of 1% (Atallah et al. 2012) or eradicating it (Nimmo-Bell, 2006). Such assumptions are valid if all infected vines are symptomatic and can therefore be rogued and replaced. If we set the undetectability and latency periods to zero to replicate such an assumption, the expected disease economic costs are approximately $4,000 per hectare over 25 years when all symptomatic vines are rogued and replaced (Strategy *I*).

We find that expanding the search for *Exposed* vines beyond the immediate neighbors leads to higher disease control costs and is not economical. For instance, Strategy I_m*NS2EW* worsens the ENPV improvement relative to the strategy of no disease control (table 2.5). The estimated ENPVs become even more negative if disease search includes two additional across-column neighbors (Strategy I_m*NS2EW2*, table 2.5). Interestingly, strategies featuring a search for *Exposed* vines beyond immediate neighbors (Strategy I_m*NS2EW* and Strategy I_m*NS2EW2*) yield lower expected half-life measures (i.e., they speed rather than delay disease diffusion) compared to the strategies testing only immediate neighbors (Strategy I_m*NS* and Strategy I_m*NSEW*). Although the identification of a larger amount of infected, nonsymptomatic vines (*Exposed*) and their removal before they become *Infective* slows disease diffusion, beyond a certain level of roguing and replanting, the effect is reversed. Grapevine roguing and replanting implies replacing infected grapevines with younger healthy ones that have shorter latency periods. Once replants get infected,

they become infective in a relatively short period, and contribute to further disease diffusion. [15]

Among age-structured spatial strategies (Strategies 13 through 18), only those targeting mature vines (Strategy I_mM-*NS* and Strategy I_mM-*NSEW*) yield positive ENPV improvements over the strategy of no disease control (table 2.5). Note that age-structured spatial control strategies (Strategies 13 through 18) perform worse than their nonage-structured (Strategies 9 and 10) counterparts. In contrast with nonspatial scenarios, structuring strategies by age in spatial scenarios reduces total revenues relative to nonage-structured strategies. This relative decrease in revenues is caused by the disease diffusion generated by those *Exposed* vines left undetected because they do not neighbor *Infective-moderate* vines in the targeted age category. Note that the channels through which structuring strategies by age affects the revenues are different in nonspatial and spatial strategies. In the nonspatial scenarios, revenue increases stem from reductions in the number of unproductive replants. In spatial scenarios, structuring disease control by age decreases revenues by lowering the level of early detection.

2.4.3 Disease management insights

Our results offer new insights to vineyard managers. Alternative disease control strategies yield different results through their different allocation of disease control effort allocation over time and space. A manager deciding when and where to control

[15] For example, we find that the final number of vines rogued and replanted under Strategy I_mNS2EW is almost twice as under Strategy I_mNS. Consequently, the vineyard age under Strategy I_mNS2EW is 3 to 7 years lower than under Strategy I_mNS (by the 25th and 50th year, respectively). As a result, the expected vineyard half-life is 1,639 months (approximately 137 years) under Strategy I_mNS2EW, compared with 2,533 months (approximately 211 years) under Strategy I_mNS.

GLRD (i.e., what age, infection, and location states to target) faces tradeoffs between the ecological benefits and drawbacks of controlling earlier, more frequently, and in a more extended neighborhood. Two types of costs incentivize vineyard managers to postpone roguing depending on their discount rates. One is the expenses incurred in testing, roguing and replacing vines, and the other is the opportunity cost of roguing an infected but still-productive vine. The latter cost consists of the forgone revenues during the period replants are unproductive. Postponing those costs has to be balanced with two types of ensuing damages, namely the continued reduction in revenues of uncontrolled infected vines and the expected economic losses due to infected vines spreading the infection to healthy ones. Our results show that it is beneficial to incur the costs of disease control earlier to avoid future damages and realize the benefits of a healthy, productive vineyard. That is, for nonspatial strategies, it is better to target younger vines in their earlier infection stages. For spatial strategies, testing the neighborhood of all symptomatic vines reduces uncertainty through early detection and control. Finally, our analysis shows that incurring virus testing costs is justified for strategies testing the immediate neighbors of symptomatic vines (Strategy I_mNSEW and Strategy I_mNS).

2.5 Sensitivity Analyses

We use the ENPVs from the simulations with alternative parameter values, together with the ENPVs calculated using the baseline parameters, to guide GLRD research investments by plant scientists, plant pathologists, and entomologists. Our sensitivity analyses deal with two critical questions. First, what parameter values make

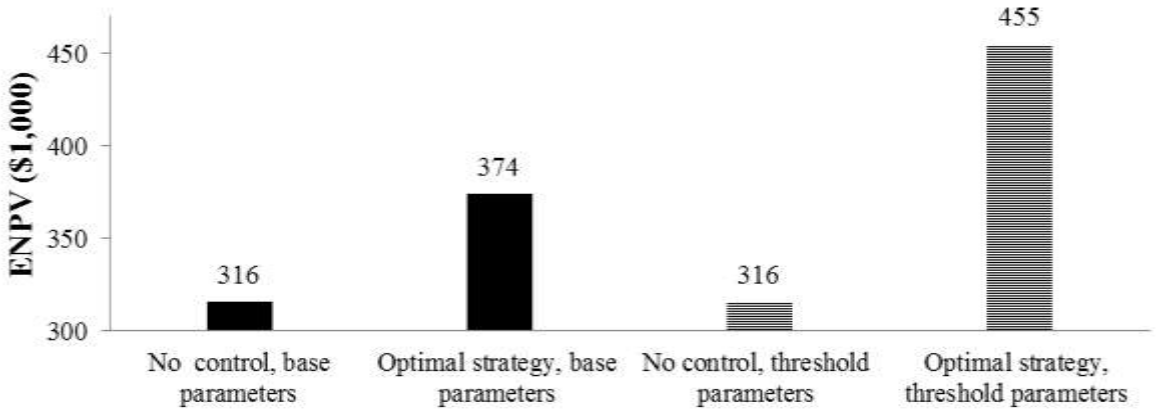
eradication possible and optimal? Second, what is the threshold expenditure for a virus-test cost beyond which the optimal spatial strategy becomes cost-ineffective?

2.5.1 Eradication feasibility and optimality

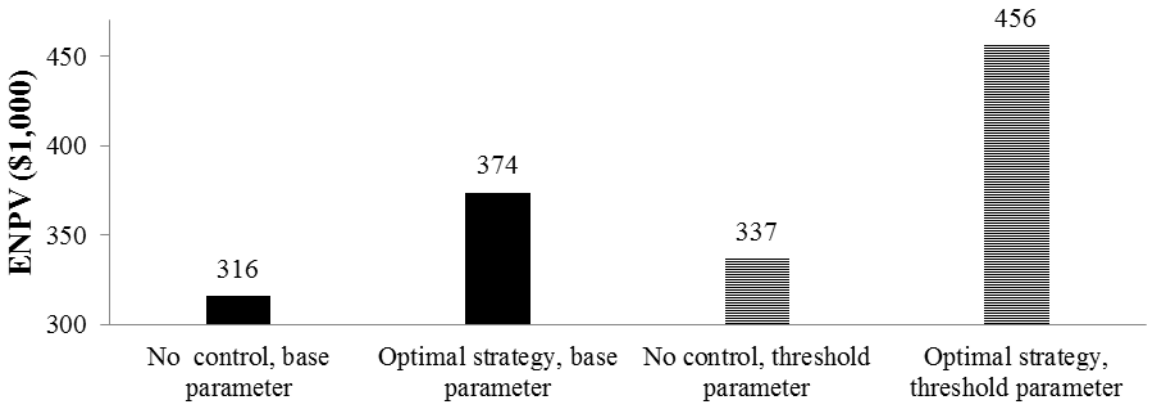
We first focus on finding parameter values that make eradication possible and optimal. We find that Strategy I_mNS and Strategy I_mNSEW achieve eradication with 99% and 100% probability, respectively, when the minimum (m_1), maximum (m_2), and mode (m_3) of the undetectability period PDF are substantially reduced. We simulate reductions from 4, 18, and 12 months in the baseline model (values in Cabaleiro and Segura 2007; Constable et al. 2012; Tsai et al. 2008) to approximately 1, 4, and 2 months, respectively (figure 2, panel a). Eradication is achieved under optimal Strategy I_mNSEW at these threshold parameter values, yielding an ENPV improvement of $139,100 (the difference between $455,100 and $316,000 in figure 2, panel a) over the strategy of no disease control. The ENPV improvement is statistically significant at the 1% level. The reduction in the undetectability period parameters might be achieved through new technology able to detect the virus one month after infection. Using the ENPVs in figure 2, we estimate the value of such technology at $81,000, under optimal Strategy I_mNSEW (the difference between $455,000 and $374,000 in figure 2, panel a).[16]

[16] Recall that our unit of analysis is a 5.2 acre vineyard with 5,720 vines.

(a)



(b)



(c)

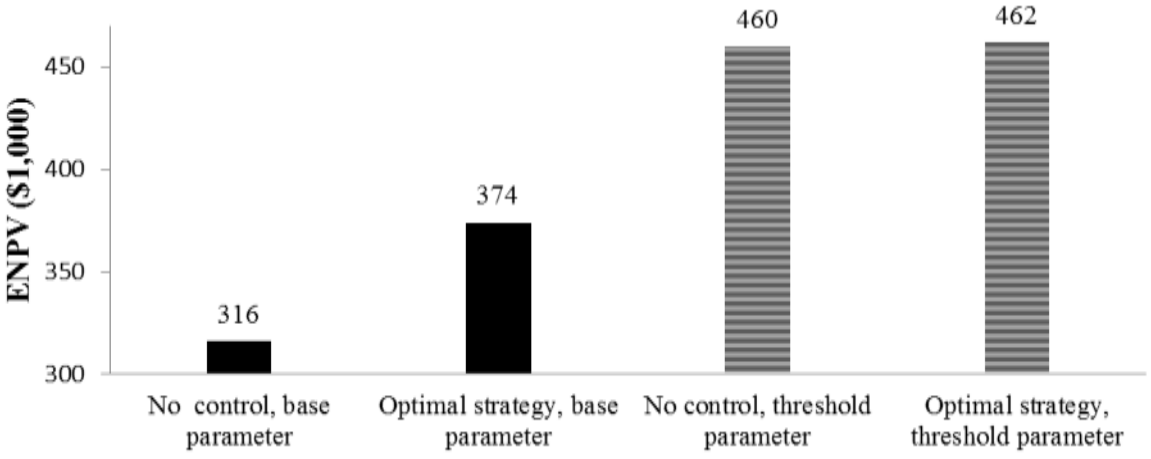


Figure 2. Sensitivity of Expected Net Present Values (ENPVs) to the Virus Undetectability Period (a), Within-Column Transmission (b), and Initial Infection Level (c) Parameters.

Eradication is also possible and optimal under Strategy I_mNS with a probability of 97% if the within-column transmission rate (α) is reduced from 4.2 to 0.1. At this threshold rate, Strategy I_mNS yields an ENPV improvement of $119,000 over the strategy of no disease control (the difference between $456,000 and $337,000 in figure 2, panel b). This ENPV improvement is statistically significant at the 1% level. Reducing the within-column transmission rate to 0.1 per month has a value of $82,000 under optimal Strategy I_mNS (the difference between $456,000 and $374,000 in figure 2, panel b). Reduction in within-column transmission is theoretically possible through vector management. However, monitoring and controlling mealybugs through biological control methods and pesticides has proven ineffective to date (Daane et al. 2012).

The initial infection level is a third critical parameter that may affect the possibility and optimality of eradication. Lowering the initial infection level from the base value (2%) to the lowest possible level (0.02% or one initially infected vine), achieves eradication with a probability of 50% under optimal Strategy I_mNSEW. At this initial infection level, Strategy I_mNSEW yields an ENPV improvement of $2,000 over the strategy of no disease control (difference between $462,000 and $460,000 in figure 2, panel c). The ENPV improvement is statistically significant at the 1% level. A research program targeting reductions of initial infection levels has a value of $88,000 under optimal Strategy I_mNSEW (difference between $462,000 and $374,000 in figure 2, panel c). For example, this result suggests that a sanitary program ensuring that 99.98% of the supplied planting material are virus-free (i.e., 0.02% initial infection level), may be justified if the cost is less than $88,000 for a 5.2 acre-vineyard

(e.g. the National Clean Plant Network of the U.S. Department of Agriculture, Johnson 2009).

2.5.2 Disease Control Costs

Finally, we find that the ENPV improvement under the second-best Strategy (I_mNS) is less sensitive to increases in disease control costs than under optimal Strategy I_mNSEW. When the virus-test cost increases twofold (from 2.6 to 5.2 $/vine), Strategy I_mNS becomes optimal. The ENPV improvement for the originally optimal, more testing-intensive Strategy I_mNSEW, decreases by 8 percentage points (from 19% to 11%, figure 3). In contrast, the ENPV improvement for Strategy I_mNS decreases by just 4 percentage points (from 18% to 14%, figure 3). Beyond a fivefold cost increase (from 2.6 to 13 $/vine), Strategy I_mNSEW becomes cost-ineffective (figure 3). We also vary the costs of roguing and replanting and find that, when the unit cost of roguing and replanting is 1.2 times higher than the base value, Strategy I_mNSEW becomes cost-ineffective. Strategy I_mNS, on the other hand, retains its cost-effectiveness up to a break-even parameter value of $30/vine (4-fold increase over the base value).

For vineyard managers, these sensitivity results highlight that, although Strategy I_mNSEW is optimal under the base parameter values, second-best Strategy I_mNS is less sensitive to changes in the costs of disease control. For scientists working on the disease, these results indicate that, although disease eradication can be optimally achieved if an early-detection technology is developed, the cost of this technology should not exceed $13/vine for it to be cost-effective in spatially controlling GLRD.

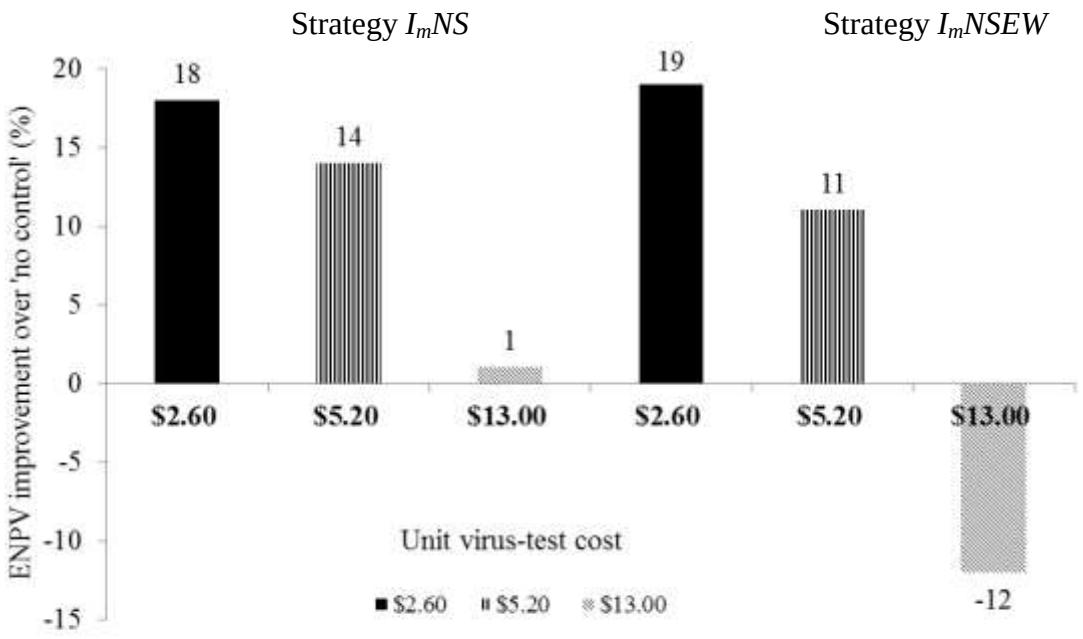


Figure 3. Sensitivity of the Expected Net Present Value to the Unit Virus-Test Cost

2.6 Conclusions and Directions for Future Research

There is growing interest in researching the economics of integrated spatial-dynamic processes in general, and pests and diseases in particular. This article features a plant-level, bioeconomic model of grapevine leafroll disease diffusion and control in a vineyard. We analyze alternative disease control strategies that would not be possible using classical approaches. The originality of the results lies in the computational method's ability to model a large number of bioeconomic, plant-level state variables.

Our results show a general feature of spatial-dynamic processes: optimal policy interventions are those that achieve the temporally, spatially, and quantitatively optimal allocation of inputs. The results are particularly valuable for vineyard managers because they highlight the superiority of spatial strategies over nonspatial

strategies recommended to the industry. We also estimate the expected value of research programs aiming at decreasing or increasing the critical model parameters. These results can help guide research efforts of disease ecologists, plant pathologists, entomologists, and plant scientists involved in GLRD research.

This model has wider application possibilities and can be adapted to other crop diseases characterized by spatial-dynamic processes after adjustments for spatial configuration and input data. In particular, it can be employed to inform profit-maximizing disease management in horticultural crops such as apple or citrus trees.

There are opportunities to extend the model as well. One extension would incorporate temporal price dynamics. If grape prices are substantially lower or higher than the mean price in the first year, optimal disease control strategies might be different than the ones identified in this article. Another extension would allow for spatial externalities caused by the flow of vectors from neighboring infected vineyards left uncontrolled. We expect this situation to yield strategies that alter the spatial configuration of the vineyard in a way that slows down disease progression. One such strategy might involve the creation of barriers to disease diffusion (e.g. Sharov and Liebhold 1998; Brown, Lynch and Zilberman 2002; Foroutan 2003). Establishing "fire breaks" from an adjacent, infected vineyard may result in immediate yield losses that will need to be measured against the expected value of lower disease damages in the future. If cost-efficient, these designs might be recommended for the establishment of more disease-resistant vineyards and orchards with higher ENPVs.

CHAPTER 3

SPATIAL-DYNAMIC EXTERNALITIES AND STRATEGIC BEHAVIOR IN INFECTIOUS DISEASE CONTROL: APPLICATION TO VINEYARDS

3.1 Introduction

Incompatible production practices within and among agricultural systems can be a source of negative externalities and potentially cause conflicts between growers. Examples of such conflicts include those that arose in California between cotton and olive growers over the spread of a plant disease (Parker 2000), between wheat and wine growers due to an herbicide drift in Washington (Corp and Darnell 2002), and between growers of genetically modified crops and conventional crops related to the dispersal of genetically modified pollen (Ceddia et al. 2011).

In these examples (disease spread, pesticide drift, pollen-mediated gene flow) conflict arises from negative externalities. That is, they are due to actions taken by growers that cause costs or damages borne by other growers. In particular, these externalities are spatial-dynamic. They are spatial because their marginal impacts decrease as distance between the source and sink of the externality increases. Moreover, these spatial impacts change over time according to the endogenous biophysical process governing the externality (e.g., inter- and intra-plot disease diffusion) and exogenously according to private and cooperative management strategies or even public policy (e.g., disease control).

A particularly interesting and policy-relevant subset of spatial-dynamic externalities in agriculture consists of those where total payoffs and cooperation to

manage the externality depend directly on the inherent heterogeneity between agents. Such situations have been examined more generally by the public economics literature by asking whether and how inherent heterogeneity affects social welfare and cooperation in the provision of public goods. This literature has examined various dimensions of heterogeneity: inequality in income, assets, and stakes; and other types of inequality such as ethnic and social, gender, and diversity in resource management technology (e.g., fishing techniques) (Baland et al. 2007). Except for income and wealth inequality, the literature suggests that all other dimensions of heterogeneity have an unambiguously negative effect on collective action. As for income or wealth inequality, public economics theory is ambiguous in predicting whether or not heterogeneity facilitates or exacerbates cooperation, especially in decentralized settings where the establishment of a regulatory authority is not possible (Gaspart and Plateau 2001). Because heterogeneity is a multidimensional phenomenon, its influence on cooperation may differ depending on the type of public good studied and on whether benefits from cooperation are measured in terms of output, surplus, or utility (Baland et al 2007). For instance, Olson (1965) hypothesizes that inequality might favor the provision of a public good by increasing the likelihood that the wealthy can provide the public good independently of the actions of others. On the other hand, Warr (1983) shows that the private provision of a public good is independent of differences in income. In contrast, Kanbur and Keen (1993) show that heterogeneity can exacerbate inefficiency from noncooperative behavior. In addition, empirical evidence from the environmental and public economics literature has found harmful effects of inequality on cooperation (e.g., Bardhan 1995). Motivated by the ambiguity

of these results, Dayton-Johnson and Bardhan (2002) propose a model that captures both the positive and harmful effects of inequality on cooperation. They use a two-player noncooperative model of conservation of a common-pool resource. They propose a U-shaped relationship between inequality and economic efficiency that is supported by empirical findings (Bardhan 2000; Khwaja 2007).

In this paper, we build on this literature to analyze the case of two neighboring wine grape growers who are heterogeneous in the wine grape quality they produce, and consequently, in the prices they receive. One grower produces high-value wine grapes and the other low-value wine grapes. They both face the spread of a grapevine viral disease that lowers grape yields and wine quality but only the high-value grower receives a price penalty for disease-affected grapes. Because of the infectious nature of the disease, the optimal disease control effort of one grower affects potential damages caused by the disease in the neighboring vineyard. We model the spatial-dynamic disease diffusion at the plant level within the vineyards using cellular automata as in Atallah et al. (2014). We also model disease diffusion between vineyards by specifying plant-level, distance- and density-dependent disease externalities. We solve for the noncooperative and cooperative disease management strategy for each grower. Subsequently, we use mean-preserving price differential contraction to show how heterogeneity between the two vineyards (i.e., the price differential) affects (a) the total payoff, (b) gains to cooperation, (c) whether cooperation is Pareto-improving, and (d) the relative magnitude of transfer payments between vineyards. We find that increased heterogeneity reduces the total payoff up to a point where cooperation

becomes Pareto-improving and the relationship between total payoff and heterogeneity becomes U-shaped.

3.2 Literature Review

Examples of conflicts arising from incompatibility in production practices include conflicts between forestry and agriculture, between growers of different crops, and even between growers of the same crop growing differentiated products. Smoke from wildfires and prescribed burning in Western Australia caused wine produced from nearby vineyards to have undesirable smoke-like flavor (Fisher, Kennison and Ward 2009). The physical drift and aerial dispersal of herbicides from wheat farmers to neighboring vineyards in the Walla Walla River Valley in Oregon and Washington states reduced foliage development and fruit set (Corp and Darnell 2002). Finally, with premiums for organic products and potential premiums for non-genetically modified (non-GM) food, recent research considers spatial policies that can remediate 'contamination' from conventional to organic crops and from GM-crops to non-GM crops (Ceddia et al. 2011).

Such policies are sometimes referred to as coexistence policies. Ceddia et al. (2011) compared the effects of several coexistence policy variables on maximizing joint grower benefits while reducing negative externalities arising from pollen-mediated gene flow from GM-crops ('externality emitters') to non-GM crops ('externality recipients'). First, they consider a mandatory buffer zone to the emitters of the externality (i.e., abatement). Second, they analyze two taxes, one imposed on the emitters of the externality (accounting for the contamination and recipient's self-

protection through a buffer area) and another on the recipient of the externality (accounting for the generator's abatement through a buffer area). Third, they consider a mechanism to incentivize both emitters and recipients to invest in coordination effort. Their simulation findings suggest that coordination to spatially cluster is most effective at reducing the externality, followed by the creation of buffer zones between GM-crops and non-GM crops. Similarly, Parker and Munroe (2007) provide empirical evidence externalities influence the location and spatial clustering of organic farms in California.

Inter-farm diffusion of pests and diseases is similar to pesticide drift and pollen-mediated gene flow in that it causes distance-dependent, spatial externalities. That is, the marginal impacts of spatial externalities decrease with the distance between emitters and recipients of the pest or disease. By contrast, however, managing externalities generated by plant pests and diseases is not limited to ex-ante (e.g., regulation) and ex-post (e.g., liability) measures as in the case of GM-crops (Demont et al., 2009; Beckman et al. 2010). Plant pests and diseases are controlled through spatio-temporal efforts exerted at fine temporal scales during the unfolding of the biophysical process responsible for the externality, thus giving it its spatial-dynamic nature. In addition to being distant dependent, the externality in the case of pests and diseases is density dependent. In other words, it depends on the location and number of the plants emitting the externality. More importantly, in the case of infectious diseases, the externality might need to be specified as a function of the infection and age states of individuals so that 'recipients' become 'emitters' after infection.

In this paper, we consider the case of Grapevine Leafroll Disease (GLRD), the most important viral disease threatening grape harvests and wine production in the United States (Fuchs et al. 2009; Golino et al. 2008; Martin et al. 2005) and around the world (Cabaleiro et al. 2008; Charles et al. 2009; Martelli and Boudon-Padieu 2006). This viral disease reduces yield, delays fruit ripening, and negatively affects wine quality by lowering soluble solids and increasing fruit juice acidity (Goheen and Cook 1959; Martinson et al. 2008). Its economic impact was recently estimated at $25,000-$40,000 per hectare if the disease is left uncontrolled, which represents more than 75% of a vineyard's net present value (Atallah et al. 2012). GLRD is primarily introduced to vineyards through infected planting material. Once introduced, the disease can be transmitted from vine to vine by several species of mealybugs and soft-scale insects (Martelli and Boudon-Padieu 2006; Pietersen 2006; Tsai et al. 2010). Mealybugs can transmit GLRD within and across vineyards in at least three ways (Charles et al. 2009; Grasswitz and James 2008). Insects crawling on wires and fruiting canes can cause disease transmission to neighboring vines. Vineyard management activities can facilitate mealybug dispersal to farther neighboring vines within the same vineyard. Additionally, disease spread between neighboring blocks or vineyards can take place through aerial dispersal of mealybugs (Le Maguet et al. 2013).

Vineyard managers are currently advised to avoid introducing GLRD into their vineyards by planting certified vines derived from virus-tested mother plants (Almeida et al. 2013; Fuchs 2007; Golino et al. 2002). However, when GLRD is already present, disease management consists mainly of minimizing the source of infection by removing symptomatic vines after harvest, especially the young ones and replacing

them with virus-tested vines (Maree et al. 2013; Rayapati, O'Neil and Walsh 2008; Walton et al. 2009). Managing disease vectors is recommended to reduce disease transmission (Skinsis et al. 2009). Although insecticide sprays can reduce mealybug densities, they have not been effective at controlling GLRD spread, mainly because of the exceptionally low insect density needed for disease transmission (Almeida et al. 2013; Cabaleiro and Segura 2006, 2007; Golino et al., 2002, 2008).

Most GLRD research has focused on studying the pathogens with less work done on disease ecology and disease management (Almeida et al 2013). Nonetheless, recent research has evaluated nonspatial and spatial GLRD control strategies in a virtual, isolated vineyard (Atallah et al. 2014). Using computational experiments, the authors show that spatial strategies improve the expected net present value of a vineyard by around 20% over the strategy of no disease control. In these strategies, young symptomatic vines are rogued and replaced, and their nonsymptomatic immediate neighbors are virus-tested, then rogued and replaced if the test is positive. It is not immediately clear, however, whether these spatial disease control strategies remain profit-maximizing in cases where disease diffusion is characterized by an "edge effect". Charles et al. (2009) refer to an "edge effect" when the number of infected grapevines is decreasing with the distance from the edge of the vineyard to the source of infection. In the case of GLRD, the edge is typically an entry point for vectors carried by wind from a neighboring vineyard serving as a GLRD reservoir (Grasswitz and James 2008). Once mealybugs enter a vineyard through aerial dispersal, they are able to move between adjacent plants. In the presence of aerial dispersal from a neighboring vineyard, roguing and replanting strategies are likely to

be less effective and additional control measures might be necessary (Rayapati et al. 2008). Some studies examining spatial diffusion of GLRD report clusters of infected vines at the borders of a vineyard. These studies measure inter-vineyard GLRD spread as a diffusion gradient, with diminishing disease incidence from the border towards the center of the vineyard (Cabaleiro and Segura 1997, 2008). In most cases, the diffusion gradient increases in the opposite direction of an adjacent, older, infected vineyard (Klaassen et al. 2011). The major environmental factor affecting long-distance mealybug aerial dispersal is wind, which has been shown to disperse mealybugs across distances greater than 50 km (Barrass, Jerie, and Ward 1994).

Inter-vineyard GLRD transmission can be depicted as a problem of transboundary renewable resource management in ecologically-connected and independently-managed systems (Munro 1979; Bhat and Huffaker 2007). In the case of agricultural diseases, differences in the value of the product can affect disease control strategies employed by growers. Using plot-level panel data, Lybbert et al. (2010) find that high-value winegrape growers make greater efforts to control powdery mildew (fungal disease) treatment strategies in response to disease forecast information more than their low-value counterparts. Fuller (2011) finds that the optimal strategy for winegrape growers is to abandon the blocks infected by Pierce's Disease (bacterial disease) if prices paid for grapes were below a certain threshold. In ecologically-connected vineyards, such difference in product value or price differential might cause conflicts in disease management strategies whereby the low-value vineyard acts as a vector reservoir of GLRD for the high-value vineyard. When vineyards are independently managed, negative spatial externalities can arise due to

the failure of one grower to internalize the negative externality of his disease management decisions on his neighbor's expected net revenues.

Traditionally, the literature on the economics of controlling insect-borne plant diseases has examined the temporal dimensions of disease control strategies (e.g. Hall and Norgaard. 1973; Regev et al. 1976). In the last decade, however, studies have concentrated on the spatial dimensions of disease control strategies. Brown et al. (2002) show that vineyard managers can maximize profits by planting barriers or by removing the source of Pierce's Disease. Their model, however, is static and largely ignores certain critical production costs such as replanting. More importantly, the authors fail to consider adjacent vineyards and incentives for cooperation in controlling the disease. More recently, Fuller et al. (2011) address the limitations of Brown et al. (2002) and show that cooperative management of Pierce's Disease can reduce grapevine losses and land abandonment. Nevertheless, their study does not identify specific cooperation mechanisms among vineyard managers.

This study contributes to the public economics literature by improving our understanding on how inherent heterogeneity affects strategic behavior and total payoff. The particular case study of an agricultural infectious disease contributes to the bioeconomics and resource economics literature by modeling the spatial and dynamic dimensions of inter-farm disease transmission at the plant-level, as opposed to using farm-level specifications (e.g., Keeling et al. 2001) or fixed diffusion rates (e.g., Fuller et al. 2011). Finally, this work extends previous research on disease management in vineyards where inter-vineyard transmission was not considered (Atallah et al. 2014).

We embed the disease diffusion model in a two-agent bargaining game to generate distributions of payoffs under all possible combinations of disease management strategies. To do this, we build on the literature on transboundary resource management that has used game-theoretic frameworks to characterize bargaining games between adjacent resource managers (Munro 1979; Sumaila 1997; Bhat and Huffaker 2004). We use the Nash bargaining framework to estimate the magnitude of the cooperative surplus and transfer payments between growers.

3.3 A Bioeconomic Model of Disease Diffusion under Spatial-Dynamic Externalities

We consider two ecologically-connected, independently-managed vineyard plots. Vineyard H produces high-value winegrapes whereas vineyard L produces low-value winegrapes. Vineyard H is represented by grid G_H that is the set of $I * J$ cells denoted by their row and column position (i, j) and representing grapevines. Similarly, Vineyard L is represented by grid G_L that consists of $M * N$ cells denoted by their row and column position (m, n). The economic problem and spatial-dynamic disease diffusion constraints faced by the managers of vineyards G_H and G_L are structurally similar and only differ in their initial conditions and bioeconomic parameters. We therefore restrict the model description to plot G_H. The disease is introduced through infected plant material to vineyard G_L , from which it spreads to vineyard G_H. Each grapevine is modeled as a cellular automaton that updates its infection state in each discrete time based on the infection state of its immediate neighbors and on long-distance disease diffusion. A vine's infection state transitions are governed by a

Markov Chain model. We first expose the growers' private profit maximization problem. Later, we expose the Nash bargaining maximization problem under cooperative disease management.

The objective of G_H's manager is to maximize the expected net present value ($ENPV_H$) of vineyard plot G_H by choosing an optimal disease control strategy from a discrete set of spatial and nonspatial strategies, $\mathcal{W}$. In each strategy, the grower decides, for each vine in cell (i,j) in each period t of T discrete periods of time, whether or not to rogue and replant ($u_{w_{i,j,t}} = 1$ if roguing takes place, 0 otherwise), test for the virus ($v_{w_{i,j,t}} = 1$ if virus testing takes place, 0 otherwise), or rogue without replanting ($z_{w_{i,j,t}} = 1$, if roguing without replanting take place, 0 otherwise).[17] The grower's disease control decisions are based on a vine's composite age-infection state $\boldsymbol{w}_{i,j,t}$. The composite age-infection state is defined as the combination of a vine's age state $\boldsymbol{a}_{i,j,t}$ and its infection state $\boldsymbol{s}_{i,j,t}$.

The optimal strategy $\mathcal{W}^*$ is the sequence of vine-level control variables $\{u_{w_{i,j,t}}, v_{w_{i,j,t}}, z_{w_{i,j,t}}\}$ that allocates disease control effort over space and time so as to yield the maximum ENPV improvement over the strategy of no disease control. Letting E be the expectation operator over the random vine-level revenue $r(w_{i,j,t})$, ρ^t the discount factor[18] at time *t* (in months) where $t \in \{0, 1, 2, \ldots, 600\}$, the objective of a vineyard manager is to maximize the ENPV as follows:

[17] 'Roguing and replanting' and 'roguing without replanting' are mutually exclusive strategies.
[18] $\rho^t = \frac{1}{(1+r)^t}$, where r is the discount rate.

$$(1)\ \max_{W} E \sum_{t\in T} \rho^{t} \sum_{(i,j)\in G} \left\{ \begin{array}{l} r_{w_{i,j,t}} * \left(1 - \sum_{\tau=0}^{\tau_{max}} u_{w_{i,j,t-\tau}}\right) * \left(1 - z_{w_{i,j,t}}\right) \\ - \sum_{\tau=0}^{\tau_{max}} \left(u_{w_{i,j,t-\tau}} * c_{u_{i,j}}\right) - \left(v_{w_{i,j,t}} * c_{v_{i,j}}\right) - \left(z_{w_{i,j,t}} * c_{z_{w_{i,j,t}}}\right) \end{array} \right\}$$

subject to:

(2) $\mathrm{E}(\boldsymbol{s}_{i,j,t+1}) = \boldsymbol{P}^{\mathrm{T}}\, \boldsymbol{s}_{i,j,t}$, which is the vine-level infection state transition equation where $\boldsymbol{P}^{\mathrm{T}}$ is the transpose of the infection state transition probability matrix $\boldsymbol{P}$.

The first expression in the curly brackets of equation (1) represents the revenue of a vine in location (i,j) and in age-infection state $\boldsymbol{w}_{i,j,t}$ at time *t*. If a vineyard manager decided to rogue and replant vine (i,j) in the last τ_{max} periods, then $u_{w_{i,j,t-\tau}}$ is equal to 1 and the revenue is pre-multiplied by zero for a period of τ_{max} until the replant bears fruit, where $\tau \in \{1,2,\ldots,\tau_{max}\}$, and $\sum_{\tau=0}^{\tau_{max}} u_{w_{i,j,t-\tau}} = \{0,1\}$.[19] If a vineyard manager decides to rogue a vine without replanting it ($z_{w_{i,j,t}} = 1$), the revenue from the cell corresponding to this vine equals zero from *t* to *T*. The second expression in the curly brackets represents the cost of roguing-and-replanting ($c_{u_{i,j}}$), the cost of testing ($c_{v_{i,j}}$), and the cost of roguing-without-replanting ($c_{z_{i,j}}$), pre-multiplied by their corresponding binary decision variables.

A vine's infection and age states map into a third dynamic state variable, its economic value, or per-vine revenue, $r_{w_{i,j,t}}$ defined as follows:

[19] This condition says that roguing and replanting in cell (i,j) cannot occur more than once in τ_{max} periods. It implies that a replant is never rogued before it bears fruit.

(3) $$r_{w_{i,j,t}} = 0 \; if \; a_{i,j,t} < \tau_{max}$$

$$= r_{S_{i,j,t}}\left(y_{S_{i,j,t=H}}, \widetilde{y_{S_{i,j,t}}}, p_{S_{i,j,t=H}}, \widetilde{p_{s_{i,j,t}}}\right) \; if \; a_{i,j,t} \geq \tau_{max}$$

(4) $$r_{w_{i,j,t}} = y_{(s_{i,j,t}=Healthy)} * \left(1 - \widetilde{y_{S_{i,j,t}}}\right) * p_{(s_{i,j,t}=Healthy)} * (1 - \widetilde{p_{s_{i,j,t}}})$$

Per-vine revenue equals zero if the vine's age $a_{i,j,t}$ is below τ_{max} (equation 3). Beyond that age, $r_{w_{i,j,t}}$ is known to the vineyard manager at time t. Nevertheless, the per-vine revenue is random for periods prior to t because it depends on the vine's infection state $\boldsymbol{s}_{i,j,t}$. This is because GLRD causes a yield reduction of $\widetilde{y_{S_{i,j,t}}}$ compared to the yield of a healthy vine ($y_{S_{i,j,t}=Healthy}$). In addition, grapes from GLRD-affected vines are subject to a price penalty $\widetilde{p_{s_{i,j,t}}}$ (equation 4) on the price paid for grapes harvested from healthy vines ($p_{S_{i,j,t}=Healthy}$). The same description applies to cells (*m*, *n*) in grid G_L (see table 3.1 for the notational differences between vineyards *H* and *L*).[20]

[20] Yield form a vine in the *Healthy* state ($y_{S_{i,j,t}=Healthy}$) is obtained by dividing per-acre yield in plot *H* (y_{d_H}) over planting density (d_{G_H}).

Table 3.1 Economic Parameters Faced by Managers of Plots G_1 and G_2

	$Plot\ G_H$		$Plot\ G_L$	
Vineyard layout				
Grid dimensions (rows*columns)	$I * J$	68*23=1,564	$M * N$	49*16=784
Grid row (vine) spacing (ft.)		4		5
Grid column spacing (ft.)		7		11
Revenue parameters				
Per-vine revenue	$r_{w_{i,j,t}}$	Random (eq. 2)	$r_{w_{m,n,t}}$	Random (eq. 2)
Grapes price ($/ton)[21]	$p_{S_{i,j,t}}$	5,058	$p_{S_{m,n,t}}$	726
Price penalty (%)	$\widetilde{p_{S_{i,j,t}}}$	70	$\widetilde{p_{S_{m,n,t}}}$	0
Yield (tons/acre)	y_{G_H}	4.5	y_{G_L}	10
Yield (tons/acre/month)		0.375		0.834
Planting density (vines/acre)	d_{G_H}	1,564	d_{G_L}	784
Yield (tons/vine/year)	$y_{i,j}$	0.0029	$y_{m,n}$	0.0128
Yield (tons/vine/month)		0.0002		0.0011
Yield reduction (%)	$\widetilde{y_{S_{i,j,t}}}$	Depends on $s_{i,j,t}$	$\widetilde{y_{S_{m,n,t}}}$	Depends on $s_{m,nj,t}$
$s = Exposed$	$\widetilde{y_{S_{i,j,t=E}}}$	30	$\widetilde{y_{S_{m,n,t=E}}}$	30
$s = Infectious, moderate$	$\widetilde{y_{S_{i,j,t=Im}}}$	50	$\widetilde{y_{S_{m,n,t=Im}}}$	50
$s = Infectious, high$	$\widetilde{y_{S_{i,j,t=I_h}}}$	75	$\widetilde{y_{S_{m,n,t=I_h}}}$	75
Cost parameters				
Roguing and replanting[22] ($/vine)	$c_{u_{i,j}}$	14.6	$c_{u_{m,n}}$	14.6
Roguing[23] ($/vine)	$c_{z_{i,j}}$	8	$c_{z_{m,n}}$	8
Testing ($/vine)	$c_{v_{i,j}}$	2.6	$c_{v_{m,n}}$	2.6
Discount factor (month $^{-1}$)[24]	ρ	0.9959	ρ	0.9959

[21] CDFA (2014)
[22] Klonsky, Karen, and Pete Livingston. 2009. Cabernet Sauvignon Vine Loss Calculator. Davis, CA: University of California
[23] Klonsky, Karen, and Pete Livingston. 2009. Cabernet Sauvignon Vine Loss Calculator. Davis, CA: University of California
[24] Equivalent to an annual discount rate of 5%

We now describe how the infection state transition probability matrix $\boldsymbol{P}$ governs disease diffusion. Vines in state *Healthy* are susceptible to infection. They get exposed to the virus with a neighborhood-dependent probability *b*. At this point, they enter a latency period during which they are nonsymptomatic and noninfective. At first, the virus population in the vine is below levels that can be detected by virus tests and the vine is in a state labeled *Exposed-undetectable*. The virus population reaches detectable levels with probability *c* (i.e., the vine transitions to state *Exposed-detectable*). The transition to state *Infective-moderate* (I_m) happens with a probability d and marks the end of the latency period and the beginning of the infectivity period as well as the onset of visual symptoms. Symptoms, which consist of reddening and downward rolling of the leaves, are at moderate severity state first (*Infective-moderate*, I_m), and subsequently transition to a state of high severity, or *Infective-high* (I_h), with a probability f. Mathematically, $\boldsymbol{P}$ can be expressed as follows:[25]

$$\boldsymbol{P} = \begin{pmatrix} (1-b) & b & 0 & 0 & 0 \\ 0 & 1-c & c & 0 & 0 \\ 0 & 0 & (1-d) & d & 0 \\ 0 & 0 & 0 & (1-f) & f \\ 0 & 0 & 0 & 0 & 1 \end{pmatrix} \tag{5}$$

In equation (5), b is the *Healthy* to *Exposed-undetectable* transition probability conditional on previous own and neighborhood infection states. It can be expressed as:

[25] $\boldsymbol{P}$ reads from row (states *H*, E_u, E_d, I_m, and I_h at time *t*) to column (states *H*, E_u, E_d, I_m, and I_h at time *t+1*).

(6)

$$b = \Pr\left(s_{i,j,t+1} = E_u \mid s_{i,j,t} = Healthy\right) =$$
$$\begin{cases} 1 - e^{-\gamma_{L,H}} \text{ if } s_{N_{i,j,t}} = (NI, NI, NI, NI) \\ 1 - e^{-(\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (NI, NI, I, NI) \\ 1 - e^{-(\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (NI, NI, NI, I) \\ 1 - e^{-(2\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (NI, NI, I, I) \\ 1 - e^{-(\alpha+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (I, NI, NI, NI) \\ 1 - e^{-(\alpha+\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (I, NI, I, NI) \\ 1 - e^{-(\alpha+\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (I, NI, NI, I) \\ 1 - e^{-(\alpha+2\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (I, NI, I, I) \\ 1 - e^{-(\alpha+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (NI, I, NI, NI) \\ 1 - e^{-(\alpha+\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (NI, I, I, NI) \\ 1 - e^{-(\alpha+\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (NI, I, NI, I) \\ 1 - e^{-(\alpha+2\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (NI, I, I, I) \\ 1 - e^{-(2\alpha+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (I, I, NI, NI) \\ 1 - e^{-(2\alpha+\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (I, I, I, NI) \\ 1 - e^{-(2\alpha+\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (I, I, NI, I) \\ 1 - e^{-(2\alpha+2\beta+\gamma_{L,H})} \text{ if } s_{N_{i,j,t}} = (I, I, I, I) \end{cases}$$

In equation (6), $s_{N_{i,j,t}}$ is the infectivity state of a vine's von Neumann neighborhood.[26] For example, $s_{N_{i,j,t}}$ = (*I*, *I*, *I*, *NI*) is the state of a neighborhood composed of two *Infective* (*I*) neighbors in the same column (within-column neighbors), one *Infective* neighbor in the adjacent column (across-column neighbor) and one *Noninfective* (*NI*) neighbor in the other adjacent column (across-column neighbor). Given that each of the four neighbors can be in one of two infectivity states

[26] This type of neighborhood represents the most common vertical trellis system where a vine is in contact with its four neighbors in the cardinal directions. In contrast, a horizontal trellis system favors contact with up to eight neighbors (Cabaleiro and Segura 2006) and could be represented by a Moore neighborhood.

(*I* or *NI*), $s_{N_{i,j,t}}$ can be in one of the 2^4 states listed in equation (6). Parameters *α, β, and* $\gamma_{L,H,t}$ are defined in the following section.

Short-distance disease diffusion

Parameters α and β are the within-column and across-column transmission rates with $\alpha > \beta > 0$, suggesting that *Infective* vines transmit the disease to their neighbors within the grid column at a higher rate than they transmit it to their neighbors situated in the adjacent grid column. We choose this neighborhood-based infection state transition to reflect patterns of GLRD diffusion observed in spatial analyses where the disease is shown to spread preferentially along grid columns (Habili et al. 1995; Le Maguet et al. 2013). We assume that within- and across-column infections occur independently with rate parameters α and β. That is, the time a vine stays in the *Healthy* state before transitioning to the *Exposed-undetectable* state is an exponentially-distributed random variable, with rate α for within-column state transitions and rate β for across-column state transitions. In each time step, a random variable u_t determines whether a *Healthy* vine transitions to the *Exposed-undetectable* state. A *Healthy* vine that has one within-column *Infective* neighbor (e.g., $s_{N_{i,j,t}} = I, NI, NI, NI$) receives the infection at time *t+1* if $u_t < 1 - e^{-\alpha}$, where u_t is a random draw from $U\sim$ (0, 1). Conversely, the disease is not transmitted if $u_t \geq 1 - e^{-\alpha}$. Similarly, a *Healthy* vine that has one across-column *Infective* neighbor (e.g., $s_{N_{i,j,t}} = NI, NI, I, NI$) receives the infection at time *t+1* if $u_t < 1 - e^{-\beta}$ and does not receive the infection if $u_t \geq 1 - e^{-\beta}$. When two or more transmission types are realized (e.g., when a vine has one *Infective* within-column

neighbor and one *Infective* across-column neighbor), the disease transmission is determined by the shortest of the waiting times (Cox 1959).[27]

Long-distance disease diffusion

Long-distance dispersal of mealybug vectors from G_L to G_H is governed by a random variable T_3, which is independently distributed with p.d.f. $\gamma_{L,H}\, e^{\gamma_{L,H}\, t}$. Here, $\gamma_{L,H,t}$ is a power-law dispersal parameter specified by the following spatial-dynamic, distance- and density-dependent dispersal function:

$$\text{(7a)}\quad \gamma_{L,H,t} = j^{-\gamma} * \frac{\sum_n^N \sum_m^M \left((m,n) \middle| s_{m,n,t} = Infective \right) * n}{\sum_n^N M * (N - n + 1)}, \qquad \gamma > 0$$

$$\text{(7b)}\quad \gamma_{H,L,t} = (N - n)^{-\gamma} * \frac{\sum_i^I \sum_j^J \left((i,j) \middle| s_{i,j,t} = Infective \right) * j}{\sum_i^j I * (J - j + 1)}, \qquad \gamma > 0$$

For any vine (i, j), $\gamma_{L,H,t}$ is inversely proportional to the distance from the shared border (i.e., column j for G_H and column $(N - n)$ for G_L).[28] We choose a power-law dispersal specification because it allows us to model the GLRD inter-vineyard transmission characteristic whereby new infection foci of infection emerge beyond the disease front (Gibson 1997; Reynolds 2011). The parameter $\gamma_{L,H,t}$ is also proportional to the total number of *Infective* vines in G_L, weighted by their column position n (numerator in equation 7a).[29] Weighting each *Infective* vine by its column position n allows vines in bordering columns to contribute more to the externality than

[27] We compare the random variable with the transition probability in each time step because the state of a vine's neighborhood is changing over time, thus changing the probability that a vine receives the infection in each time interval.

[28] For the power-law dispersal parameter γ, we use the estimated slope ($\gamma_{=3}$) of the disease gradient obtained by regressing the natural logarithm of GLRD incidence on the natural logarithm of the distance (column position) in the Sisan vineyard plot in table 1 of Cabaleiro and Segura (1997).

vines situated farther from the border (i.e., distance-dependence).The denominator in equation (7a) allows the multiplier of the power-law expression (the term in the double summation) to vary between 0 and 1 as the number of *Infective* vines in G_L varies between 0 and $M * N$. Once vines in vineyard G_H become *Infective*, they can act a source of infection for *Healthy* vines in vineyard G_L according to equation (7b), thus making the externality reciprocal.[30]

When more than one type of disease transmission occurs, such as when a vine has one *Infective* within-column neighbor (short-distance disease diffusion) and is situated on the border of the vineyard (long-distance disease diffusion), the realized type of transmission is determined by the smaller value among T_1, T_2 and T_3 (Cox 1959). For descriptions of probabilities *c*, *d*, and *f*, we refer the reader to Atallah et al. (2014). Symbols, definitions, values, and references for the disease diffusion parameters are presented in table 2.1.

[30] In situations where the externality needs to be asymmetrical (e.g., because of prevailing winds), γ can be given different values in equations 7a and 7b. Note that a value of zero in bot equations collapses the model to the one in Atallah et al. (2014).

3.4 Experimental Design

We design and implement Monte Carlo experiments to evaluate various disease control strategies by comparing their economic outcomes to those resulting from a *no disease control* strategy. Each experiment consists of a set of 1,000 simulations run simultaneously for both vineyard plots, over 600 months. Experiments differ in the disease control strategies employ. Outcome realizations for a given run within an experiment differ due to random spatial initialization in G_L, and due to random spatial disease diffusion within and between vineyards. Data collected over simulation runs are the probability density functions of the ENPVs under each strategy.

3.4.1 Model initialization

Grapevines are initialized as *Healthy* and of age equal zero in both vineyard plots G_H and G_L (high- and low-value vineyard, respectively). At $t = 1,$ one percent of the grapevines in G_L are chosen at random from a uniform spatial distribution U (0, $M * N$) to transition from *Healthy* to *Exposed* (recall that $M * N$ is the total number of grapevines in G_L where M and N are the number of rows and columns, respectively). This reflects findings in GLRD studies indicating that primary infection sources are randomly spatially distributed (Cabaleiro et al. 2008), and that initial disease prevalence is typically between one and five percent (Atallah et al. 2012). Subsequently, GLRD spreads to *Healthy* vines within G_L according to the infection-state transition Markov Chain process in equation (4). The *Infective* vines in G_L act as a primary source of long-distance disease diffusion to the *Healthy* vines in G_H. The disease spreads from G_L to G_H according to the distance- and density-dependent dispersal function $\gamma_{L,H}$ (equation 7a). Subsequently, *Infective* vines in G_H act as a

source of reinfection in G_L according to the distance- and density-dependent dispersal function $\gamma_{H,L}$ (equation 7b). Symbols, definitions, values, and references for the economic parameters (e.g., prices, quality penalty, and disease control costs) are presented in table 3.1.

3.4.2 Disease control strategies

The discrete set of disease control strategies, $\mathcal{W}$, includes three subsets, namely nonspatial, spatial, and fire-break strategies. In the subset of *nonspatial strategies*, the vineyard manager decides whether to rogue and replace symptomatic vines based on their symptoms (*Infective-moderate; Infective-high*) with or without considering their age (*Young*: 0-5 years; *Mature*: 6-19 years; *Old*: 20 years and above). There are eight nonspatial strategies (Strategy 1 to Strategy 8 in table 2.4).[31] In the subset of *spatial strategies*, the vineyard manager decides whether to rogue and replant vines as soon as they develop symptoms (*Infective-moderate*), test their neighbors and rogue-and-replace them if they test positive. If a vine tests positive, it is removed in the same period. We simulate 10 spatial strategies (Strategy 9 to Strategy 18 in table 2.4). Note that, when implementing nonspatial or spatial strategies, a manager's disease risk depends heavily on whether the neighboring vineyard's manager controls for the disease or not. In these strategies, disease control in one vineyard is a strategic complement to disease control in the neighboring vineyard.

The third subset includes *fire-break strategies* that consist in roguing (without replanting) vines in the border columns of a vineyard in order to create 'fire-breaks' or

[31] We exclude the strategy of roguing and replacing *Infective-high* and *Young* (I_hY) because this age-infection combination cannot be reached; it takes a vine more than 5 years to transition to the *Infective-high* state.

'buffer zones' that would reduce long-distance disease diffusion from to G_L to G_H (Strategy 19 to Strategy 25 in table 3.2). Fire-break strategies are intended to decrease the effect of spillovers between vineyards and can give a manager full control over their disease risk. In these strategies, disease control in one vineyard is a strategic substitute to disease control in the neighboring vineyard.

The scenario of no disease provides a baseline to compute the expected disease economic cost under each candidate control strategy (Strategies 1-25). All strategies are available to both growers.

Table 3.2 'Fire-break' Disease Control Strategies: definitions and acronyms

#	Description	Acronym
19	Roguing (without replacing) all the vines in the bordering column in G_L.	*1Col*
20	Roguing (without replacing) all the vines in two bordering columns in G_L.	*2Col*
21	Roguing (without replacing) all the vines in three bordering columns in G_L.	*3Col*
…	…	…
25	Roguing (without replacing) all the vines in all *M* columns in G_L (remove the entire vineyard).	*16Col*

[a] Strategies are assumed to be implemented at $t = 24$, which corresponds to the moment when initially infected vines in G_L develop visual leafroll symptoms.

3.5 Solution Frameworks

We employ the objective function (equation 1) to rank the vineyard expected net present values under the alternative strategies for an individual vineyard. The objective function takes into account the total amount of control effort exerted under each strategy as well as the timing, intensity and location of the control. We first solve the social planner problem. The solution to this problem is relevant for situations where vineyard management companies manage contiguous vineyards that produce

different qualities of wine grapes. Second, we solve for the noncooperative solution. Third, we search for the cooperative solution using the Nash bargaining framework whenever the cooperative surplus is strictly positive.

3.5.1 Social planner

The social planner chooses the optimal pair of disease management strategies (w_H, w_L) that maximizes the total payoff given by $ENPV_T$, the sum of the expected net present values of G_L ($ENPV_L$) and G_H ($ENPV_H$). The social planner solves the following maximization problem:

(8a) $\max_{(w_H, w_L)} ENPV_H + ENPV_L$, subject to:

(2) $\mathrm{E}(\boldsymbol{s}_{i,j,t+1}) = \boldsymbol{P}^{\mathrm{T}} \boldsymbol{s}_{i,j,t}$, and

(8b) $\mathrm{E}(\boldsymbol{s}_{m,n,t+1}) = \boldsymbol{P}^{\mathrm{T}} \boldsymbol{s}_{m,n,t}$

where equations (2) and (8d) are the vine-level infection state transition equations in G_H and G_L, respectively.

3.5.2 Noncooperative disease control

In this case each vineyard manager solves his private ENPV maximization problem subject to disease diffusion in his vineyard and the disease externality from the neighboring vineyard by choosing one of the eighteen nonspatial and spatial strategies available to him (Strategies 1 through 18 in table 2.4). Because the disease is initialized in vineyard G_L, we first solve for the optimal strategies of G_L's manager. Then, given optimal disease control in G_L, the manager of G_H solves for his optimal control strategy.

3.5.3 Cooperative disease control: Nash bargaining game

The expected payoffs of noncooperative strategies constitute the players' threat points in a cooperative setting (Nash 1953). If the two vineyards are cooperatively managed, the two managers solve the Nash bargaining game to find payoffs that ensure the existence of a mutually beneficial agreement. The Nash bargaining solution is the unique pair of cooperative payoffs ($ENPV_H^C, ENPV_L^C$) that solves the following maximization problem (Nash 1953):

(9) $$\max_{\{ENPV_H,\ ENPV_L\}} (ENPV_H^C - ENPV_H^{NC})\ (ENPV_L^C - ENPV_L^{NC}),$$

subject to:

(10) $$ENPV^C \geq ENPV^{NC}$$

and subject to the disease diffusion functions in G_H (equation 2) and G_L (equation 8b). Equation (10) is the incentive compatibility constraint, which guarantees that players have incentives to cooperate and do not resort to their credible noncooperative threat with payoffs $ENPV^{NC}$. The maximand in equation (9), known as the Nash product, is the product of the differences between the cooperative and noncooperative payoffs from G_H and G_L. Under the standard axiomatic bargaining theory, equation (9) has the following unique solution (Muthoo 1999):[32]

(11) $$ENPV_H^C = ENPV_H^{NC} + \frac{1}{2}\ (ENPV_T^C - ENPV_T^{NC})$$

(12) $$ENPV_L^C = ENPV_L^{NC} + \frac{1}{2}\ (ENPV_T^C - ENPV_T^{NC})$$

[32] The axioms are individual rationality, invariance to equivalent utility representations, symmetry, and independence of irrelevant alternatives.

In the solution described by equations (11) and (12), the growers agree that each gets his expected noncooperative payoff first (i.e., the payoff of the threat point, or the disagreement point). Subsequently, they split the expected cooperative surplus equally. The latter is defined as the difference between the total expected cooperative payoff ($ENPV_T^C = ENPV_H^C + ENPV_L^C$) and the total expected noncooperative payoff ($ENPV_T^{NC} = ENPV_H^{NC} + ENPV_L^{NC}$). The expected cooperative surplus is also a measure of the Pareto-inefficiency caused by noncooperative disease control.

We also solve a cooperative game that features alternating offers through an infinite time horizon (Rubenstein 1982) and obtain the same solution as in the Nash bargaining game. Both bargaining games have the same optimal solution because our players have the same discount rate. In situations where players have different discount rates, the cooperative surplus is shared proportionally so that more 'patient' players get a higher share of the surplus.[33]

3.6 Results and Discussion

We find that, under the baseline case, both managers find it optimal not to control the disease in a noncooperative setting. The optimal solution is cooperative and consists of the high-value manager paying the low-value manager to spatially control the disease. We find that the relationship between heterogeneity, as expressed by the price differential, and social welfare, as measured by total payoff, is U-shaped.

[33] Our result is also a special case of the solution to the generalized (or asymmetric) Nash bargaining game where players have the same 'bargaining power' (Muthoo 1999, p. 35). Muthoo (1999, p. 52) also shows that the bargaining outcome generated by the Nash bargaining game is identical to the outcome generated by the basic alternating-offers model (Rubenstein 1982) when bargaining costs are small.

3.6.1 Social planner

Simulation results indicate that, under the baseline prices (p_H=$5,058/ton; p_L=$726/ton) , the total payoff is highest ($364,000/acre) when the disease is managed in both vineyards under the spatial strategy that targets symptomatic vines and their two immediate, within-column neighbors (Strategy I_mNS) (table 3.3). The ENPV improvement of this strategy over a strategy of no control is $60,000/acre, or approximately 20%. This is similar to the 18% ENPV improvement in Atallah et al. (2014) in the context of a vineyard with no externalities.

Table 3.3 Expected Payoffs under the Social Planner, Noncooperative, and Cooperative Solutions (baseline prices p_H=$5,058/ton and p_L=$726/ton)

	Expected Payoffs [a] *($1,000/acre over 50 years)*						
Strategies (G_1, G_2)	Payoff to G_H	Payoff to G_L	Total payoff	Cooperative Surplus [b]	Fixed Transfer Payment to G_L [c]	Cooperative Payoff to G_H	Cooperative Payoff to G_L
Social planner							
I_mNS, I_mNS	305 (4)	59 (6)	364	n/a	n/a	n/a	n/a
Noncooperative							
no control, no control	243 (5)[d]	62 (1)	305	n/a	n/a	n/a	n/a
Cooperative							
I_mNS, I_mNS	305 (4)	59 (6)	364	60***	32	273	91
no, Col16	362 (1)	-6 (0)	356	51***	93	269	87

n/a is not applicable.
[a] Expectations are obtained from 1,000 simulations.
[b] Cooperative Surplus= Total payoff (Cooperative)-Total payoff (Noncooperative)
[c] Fixed Transfer Payment =Expected Payoff (Noncooperative)–Expected Payoff (Cooperative)+50%*Expected Cooperative surplus.
[d] Standard deviations in parentheses.
*** Significantly different from zero at the 1% level.

3.6.2 Noncooperative disease control

In a noncooperative setting, the manager of the low-value vineyard G_L finds it optimal not to control the disease. In this case his vineyard's ENPV is $62,000/acre (table 3.3).

G_L's second-best strategy is I_mNS, which yields and ENPV of \$59,000/acre (not shown in table 3.3).[34] Given G_L's decision of not controlling the disease, G_H's optimal response is not to control the disease either, yielding an ENPV of about \$243,000/acre. The strategy of 'no control' is therefore a credible threat point for G_L in a cooperative game. Intuitively, the optimality of 'no control' for G_H depends on the magnitude of the externality (parameter γ in equation 7a): the value of γ is such that, no matter what G_H does to control the disease, if G_L does not practice some disease control to reduce the externality, G_H will be better off doing nothing. G_H's second-best strategy is nonspatial Strategy I_mY (\$222,000/acre) (not shown in table 3.3). The optimal noncooperative expected payoffs for growers G_H and G_L are \$243,000 and \$62,000, respectively (table 3.3).

The solution to the social planner's problem indicates that, if the two vineyard managers cooperate and agree to implement spatial Strategy I_mNS [35] in their respective vineyards, then the total payoff ($ENPV_T$) would be 20% higher than in the noncooperative setting. These benefits to cooperation are consistent with previous studies on cooperative harvesting in fisheries (Sumaila 1997) and nuisance wildlife species (Bhat and Huffaker 2007). However, strategy I_mNS makes G_L worse off relative to the strategy of no disease control. In this case, the two growers could enter into a self-enforcing cooperative agreement that includes side payments negotiated ex-

[34] Recall that these strategies consist of roguing-and-replanting the moderately infected and young vines (I_mY); roguing-and-replanting the symptomatic vines, testing their two nonsymptomatic neighbors and roguing-and-replanting them if they test positive (I_mNS).
[35] Recall that this strategy consists of roguing and replacing symptomatic vines (I_m) then testing their nonsymptomatic immediate neighbors (NS) and roguing them if they test positive.

ante and threats by each vineyard manager to revert to noncooperative behavior if a party breaches the agreement.

3.6.3 Cooperative disease control

A cooperative disease management game unfolds as follows. At the beginning of the game, the vineyard managers negotiate a Pareto-efficient disease management agreement that includes a single lump-sum side payment made by the manager who benefits from cooperation to the manager that loses in a cooperative setting. The managers monitor the evolution of the game, ensuring that cooperative disease management is in place and that payments are made on the agreed-upon schedule. Growers continue cooperating unless one of them violates the agreement. If either one manager fails to pay or the other fails to implement the cooperative disease management strategy, the other party reverts to their credible threat strategy of no disease control. In order for the cooperative disease management strategy to be Pareto-efficient, the manager who gains from cooperation compensates the other manager for the difference between his noncooperative and cooperative expected payoffs. In addition, the managers get equal share of the total benefits resulting from cooperation.

According to this solution framework, the size of the fixed transfer payment from G_H to G_L is \$32,000/acre (table 3.3). After this transfer, both managers are better off than under the noncooperative setting (\$273,000 vs. \$243,000 for G_H and \$91,000 vs. \$59,000 for $G_{L,}$ table 3.3). The cooperative surplus is \$60,000/acre and is statistically significant at the 1% level.

This set of spatial control strategies assumes that enforcement costs are negligible for both growers. However, disease monitoring costs might be high and thus cause the optimal roguing-and-replanting strategy to yield lower total expected payoffs than alternative strategies. Monitoring would involve G_H observing whether G_L is identifying symptomatic vines, roguing-and-replacing them, and testing-and-roguing their immediate nonsymptomatic neighbors. In contrast, the buffer or 'fire-break' cooperative strategies are likely to have low monitoring costs for G_H (Strategies 19 to 25, table 2.4). These strategies do not require the monitoring needed in the spatial control strategies (visual identification of GLRD symptoms, testing, replanting). They only require G_H to verify that G_L has removed the bordering vineyard rows. In situations where disease monitoring costs are too high for cooperative Strategy (I_mNS, I_mNS) to remain optimal, Strategy (*no control, 16Col*) yields the highest ENPV improvement among the fire-break strategies. According to this strategy, G_H pays G_L to remove all his vines (all sixteen columns in grid G_L), in which case G_H does not need to control any disease. The cooperative surplus under this strategy is $51,000, which is statistically different from zero at the 1% level (table 3.3). The difference in cooperative surplus between cooperative Strategy (*no control, 16Col*) and cooperative Strategy (I_mNS, I_mNS) is $9,000. Disease monitoring costs would need to be higher than this difference before fire-break Strategy (*no control, 16Col*) is preferred to the spatial Strategy (I_mNS, I_mNS). In addition, note that although monitoring costs would be lower under this fire-break strategy, the higher relative magnitude of the required transfer payment compared to the cooperative surplus suggest that cooperation might be harder to achieve under this strategy.

3.6.4 Effect of the spatial price differential on strategic behavior and total payoff

In order to measure how the price differential between vineyards affects their strategic decision to cooperate, we solve the problem for six additional price pairs under all three solution frameworks. Starting with the baseline price pair (case 6, table 3.4), we conduct five mean-preserving price differential contractions (cases 1 to 5, table3. 4) and one mean-preserving price differential expansion (case 7, table 3.4). Note that the price differential is equal to zero in case 1 and it increases as we move to case 7.

Results in table 3.4 show that the price differential has a substantial influence on the managers' strategic behavior and their payoffs. These results can be discussed in terms of two distinct management situations. First, in cases 1 through 5, the high values of p_L justify the implementation of Strategy I_mNS in vineyard block G_L. In a noncooperative setting, the disease externality is reduced enough that 'no control' is justified in vineyard block G_H. The optimal noncooperative outcome is therefore (*no control*, I_mNS). In these five cases, although the cooperative surplus is strictly positive and significantly different from zero at the 1% level, it is too small to satisfy the incentive compatibility constraint of G_H (equation 10) and cooperation does not take place. If the two vineyards were managed by a single entity, they would both be managed according to Strategy I_mNS and total payoff would be higher by $200 to 2,000/acre (table 3.4, cases 1 through 5).

The second situation is given in cases 6 and 7 where the low values of p_L do not justify the implementation of any disease control in vineyard block G_L. In a noncooperative setting, subjected to the uncontrolled externality, G_H finds it optimal not to control the disease either. The optimal noncooperative outcome is therefore (*no*

Table 3.4 Impact of Price Differential on Cooperative Surplus and Transfer Payments: Price Mean-Preserving Contraction and Expansion

	Prices ($/ton)[a]		*Setting*	*Optimal strategy*		*Expected payoffs, surplus, and transfers ($1,000/acre)*						
Cases	p_H	p_L		G_H	G_L	$ENPV_H$	$ENPV_L$	$ENPV_T$	Surplus[b]	Payment	$ENPV_H$*	$ENPV_L$*
1	2892	2892	Noncooperative	no control	I_mNS	177	308	485	N/A	N/A	177	308
			Cooperative	I_mNS	I_mNS	165	321	486	1.2***	0	N/A	N/A
2	3325	2459	Noncooperative	no control	I_mNS	204	257	461	N/A	N/A	204	257
			Cooperative	I_mNS	I_mNS	193	269	462	0.6***	0	N/A	N/A
3	3758	2026	Noncooperative	no control	I_mNS	230	206	436	N/A	N/A	230	206
			Cooperative	I_mNS	I_mNS	221	215	436	0.4***	0	N/A	N/A
4	4192	1592	Noncooperative	no control	I_mNS	256	155	411	N/A	N/A	256	155
			Cooperative	I_mNS	I_mNS	249	164	413	1.7***	0	N/A	N/A
5	4625	1159	Noncooperative	no control	I_mNS	283	104	387	N/A	N/A	N/A	N/A
			Cooperative	I_mNS	I_mNS	277	111	388	0.7***	0	283	104
6	5058	726	Noncooperative	no control	no control	243	62	305	N/A	N/A	N/A	N/A
			Cooperative	I_mNS	I_mNS	305	59	364	59.8***	32	273	91
7	5491	293	Noncooperative	no control	no control	264	25	289	N/A	N/A	N/A	N/A
			Cooperative	no control	*Col16*	393	-6	387	98.2***	80	313	74

N/A is not applicable.

[a] Recall that prices in cases 1 through 6, and prices in Case 7 are obtained through a mean-preserving contraction and a mean-preserving expansion of prices in the baseline case (case 6), respectively.

[b] Recall that the cooperative surplus is defined as the difference between $ENPV_T$ (cooperative) and $ENPV_T$ (noncooperative).

*** Significantly different from zero at the 1% level.

control, *no control*). The price differential in cases 6 and 7 is sufficiently large that it favors cooperative disease management. Case 6 is the baseline case that we discussed in detail in the previous section. That is, G_H pays G_L to spatially control the disease according to Strategy I_mNS, in which case he implements the same strategy. In case 7, the price differential and cooperative surplus are even larger that G_H pays G_L to remove all 16 columns without replacement (Strategy *16col*), i.e. exit production.

Between cases 1 and 6, total payoff is monotonically decreasing in the level of heterogeneity (i.e., the magnitude of price differential) (figure 4). Cases 6 and 7 on the other hand represent a range where the relationship between price differential (more generally, heterogeneity) and total payoff (more generally, social welfare) becomes U-shaped. These are also the two cases that yield a cooperative solution. Note, however, that although cooperation becomes Pareto-improving in this range, it also becomes harder to achieve as the level of heterogeneity increases. One indicator of the difficulty of cooperation is the ratio of transfer payment to cooperative surplus, with higher ratios implying more difficult cooperation. This ratio would be 0.5 in case 6 and 0.8 in case 7 (ratios 32/60 for case 6 and 80/98 for case 7, table 3.4).

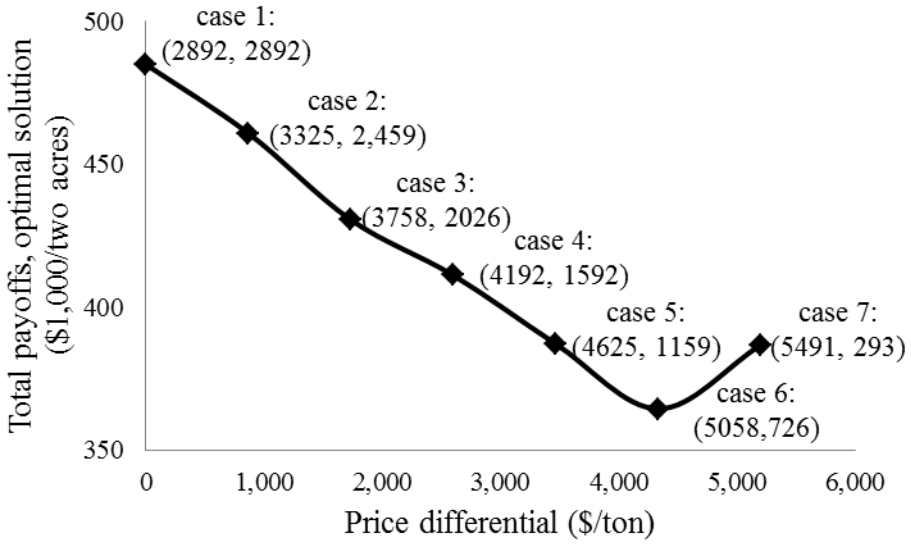


Figure 4. Total payoff under the optimal solution for each of the seven price differential cases (price pairs (p_H, p_L) in $/ton, in parentheses).

3.7 Conclusions and Next Steps

There is growing interest in the economics of integrated spatial-dynamic processes under the presence of negative externalities due to incompatible production practices in agriculture. In this paper, we develop a two-agent bargaining game in the context of a spatial-dynamic model of disease diffusion and control. We apply this model to the GLRD spread in wine grape vineyards and generate distributions of vineyard ENPVs to solve the game. Under the baseline case, if managers do not cooperate, each finds it optimal not to control the disease. The optimal solution is cooperative and consists of the high-value vineyard manager spatially controlling the disease and paying the low-value vineyard manager to employ the same disease control strategy. Using a mean-preserving price differential contraction, we find that cooperation is not optimal for smaller magnitudes of the price differential. More importantly, we find that the relationship between heterogeneity, as expressed by the price differential, and social

welfare, as measured by total payoff, is U-shaped as hypothesized by Dayton-Johnson and Bardhan (2002). In addition, we find that in the price range where greater heterogeneity increases social welfare and cooperation is optimal, the ratio of transfers to cooperative surplus is higher thus causing the cooperative solution to be harder to implement.

Our model is rich in modeling the vine-level heterogeneity in the disease diffusion process generating the externality but it is limited in its ability to model more than two agents with greater dimensions of heterogeneity that can affect total payoffs. In addition, the Nash bargaining framework used does not allow us to accommodate changing circumstances that can shift the bargaining power and the incentives to cooperate for the players over the lifecycle of their vineyards. Future research can model the problem in an agent-based computational model framework that allows for additional agents with abilities to learn over time and with greater dimensions of heterogeneity such as risk attitudes. This is especially relevant given the high level of price variability observed in the winegrape industry and the impact it can have on incentives to cooperate as well as on the size of the payments. More generally, such a framework would add to our limited understanding of collective action among heterogeneous players in a heterogeneous biophysical setting.

CONCLUSION

This dissertation provides an economic analysis of Grapevine Leafroll Disease, the most widespread viral disease in vineyards worldwide. It documents the economic cost of the disease at the vineyard level and recommends optimal management strategies with and without disease externalities. We contribute to the literature that employs nonspatial, compartmental models when modeling diseases by relaxing the simplifying assumptions that individual plants are homogenous in their attributes and spatially perfectly-mixed. We also contribute to the spatial literature by modeling spatial heterogeneity as dynamically determined by disease diffusion and control rather than specifying it as exogenous and fixed over time. We use a continuum of models, starting with a nonspatial, nondynamic economic analysis of GLRD in a single vineyard and ending with a fully spatial-dynamic model of disease diffusion and control within and between neighboring vineyards. We show that analyses that do not incorporate disease ecology parameters at the plant level in a spatial-dynamic way might not capture the imperfect information in disease diffusion and disease control and could therefore lead to suboptimal management recommendations. This dissertation also contributes to the public economics literature by improving our understanding on how inherent heterogeneity affects strategic behavior and total payoffs. The particular case study of an agricultural infectious disease contributes to the environmental and resource economics literature by modeling long-distance

dispersal at the plant-level, in a spatial-dynamic way as opposed to using farm-level specifications or fixed diffusion rates.

In the first essay, we find that the economic impact of GLRD can represent more than 75% of a vineyard's net present value if left uncontrolled. We underline the value of preventing GLRD through the use of virus-tested vines. Although nonspatial and nondynamic, the analysis in this essay is essential as it identifies the threshold initial disease prevalence beyond which vineyard replacement is economically superior to any disease control.

Essay 2 starts by laying out the unique characteristics of insect-transmitted plant diseases such as GLRD that call for developing spatial bioeconomic simulation models incorporating disease ecology parameters at the plant level. The cellular automata model developed in this essay shows that failure to model imperfect information in disease diffusion and disease control through the incorporation of disease latency and undetectability period parameters can lead to an underestimation of the disease economic cost and the unrealistic prediction of disease eradication. Most importantly, this essay demonstrates that current nonspatial strategies recommended to the industry might not be profit-maximizing and highlights the superiority of spatial strategies. The originality of the results lies in the computational method's ability to model a large number of bioeconomic, plant-level state variables. The essay also illustrates how the model can be used to prioritize GLRD research through the identification of the most critical bioeconomic model parameters.

Essay 3 examines how optimal disease management in a single vineyard can be compromised by the presence of spatial-dynamic, disease externalities and

evaluates the ability of bargaining and transfer payments to control these externalities. It does so by developing a model that embeds a computational model of disease diffusion within and between vineyards in a bargaining game-theoretic model of disease control. We find that, if neighboring managers do not cooperate, each finds it optimal not to control the disease. We compute the benefits to cooperation and show that the cooperative solution includes transfer payments and consists of each manager implementing the optimal spatial strategy outlined in Essay 2. The essay concludes with an examination of the relationship between agent heterogeneity, as expressed by the spatial price differential between the neighboring managers, and total payoff. We find that increased heterogeneity reduces the total payoff up to a point where cooperation becomes Pareto-improving and the relationship between heterogeneity and total payoff becomes U-shaped.

This dissertation highlights the value of conducting bioeconomic analyses at the individual plant level, in a spatial-dynamic way when studying plant pests and diseases that are similar to GLRD. It offers a modeling approach that can be used to examine the bioeconomics of other crop diseases characterized by spatial-dynamic processes. We hope that this dissertation will encourage applications of relatively new modeling tools and computational methods in this field. In view of the increased understanding of spatial-dynamic ecological processes and the recent developments in the spatial resource economics, it is likely that models of the sort developed in this dissertation will be essential to formulate agricultural, resource, and environmental management recommendations.

APPENDIX

Survey Instrument

1. **Prevalence:** What percentage of your vineyard is affected by leafroll? (*Highlight one*)
   a. 0
   b. 0-10
   c. 10-25
   d. 25-50
   e. 50 or more
   f. I don't know
2. **Varieties**: What grape varieties were affected by leafroll?
3. **Symptoms of Leafroll Virus on Crop:** In the following section, please mention whether you noticed a change in **per vine** yield, sugars and/or acidity associated with the leafroll virus infection. If changed occurred, please indicate the degree of change, if measured or estimated (*highlight answer*).

Yield decreased by (%):
   a. 0 (no decrease)
   b. 0-10
   c. 10-25
   d. 25-50
   e. 50 or more
   f. I don't know

Sugars decreased by (º brix):
   a. 0 (no decrease)
   b. 0-1
   c. 1-2
   d. 2-3
   e. 3-4
   f. I don't know

Acidity increased by (g/L):
   a. 0 (no decrease)
   b. 0-0.5
   c. 0.5-1
   d. 1-2
   e. 2 or more
   f. I don't know

4. **Changes In Agricultural Practices In Response To Leafroll Incidence:**
   - Have you replanted your vineyard in response to leafroll infection? Yes No (*highlight one*).
   - If not, have you changed any of the following practices as a response to your vineyard leafroll infection? (*Tick the appropriate cell*). If yes, please mention how many units (of labor or equipment) you had to utilize on each activity as a result of leafroll infection

	Yes	No	If yes, how many units (vines replanted, quantity fertilizer/pesticide, etc.)
Vine replacement			
Leaf removal			
Fertilization			
Pesticide			
Other:			

5. Contracts With Vineyards:

- Do you buy/sell grapes from vineyards other than your own? Yes No (*highlight one*).
- If yes, do you have contracts with those vineyards? Yes No (*highlight one*).
- If yes, does the contract refer to quality standards related to the sugars and/or the acidity of the grapes? Yes No (*highlight one*).
- If yes, what are those standards? Sugars:_____ Acidity:_____
- How do you penalize (get penalized for) lower standards?
  a. No penalty for lower standards
  b. Batch is refused
  c. There is a penalty of: _____

REFERENCES

Almeida, R. P., Daane, K. M., Bell, V. A., Blaisdell, G. K., Cooper, M. L., Herrbach, E., and G. Pietersen. 2013. "Ecology and management of grapevine leafroll disease." *Frontiers in Microbiology* 4:94

Atallah, Shady S., Miguel I. Gómez, Jon M. Conrad and Jan P. Nyrop. 2014. "A Plant-level, Spatial, Bioeconomic Model of Plant Disease Diffusion and Control: Grapevine Leafroll Disease." doi: 10.1093/ajae/aau032, *American Journal of Agricultural Economics*.

Atallah, S.S., M. I. Gómez, M. F. Fuchs, and T. E. Martinson. 2012. "Economic Impact of Grapevine Leafroll Disease on Vitis vinifera cv. Cabernet franc in Finger Lakes Vineyards of New York." *American Journal of Enology and Viticulture* 63:73-79

Atallah S., M. Gómez, M. Fuchs, T. Martinson and G., White 2010. "Economic impact of the grape leafroll virus (GLRV) in the Finger Lakes region of New York." *Extension Bulletin No. EB-2010-15*, Charles H. Dyson School of Applied Economics and Management, Cornell University.

Babcock, B. A., E. Lichtenberg, D. Zilberman. 1992. "Impact of Damage Control and Quality of Output: Estimating Pest Control Effectiveness." *American Journal of Agricultural Economics* 74: 163-172.

Baland, J. M., Bardhan, P. K., and Bowles, S. (Eds.). 2007. *Inequality, cooperation, and environmental sustainability*. Princeton University Press.

Balmann, A. 1997. “Farm-Based Modelling of Regional Structural Change: A Cellular Automata Approach.” *European Review of Agricultural Economics* 24: 85-108.

Bardhan, P. K. 1995. “Rational fools and cooperation in a poor hydraulic economy”, in (K. Basu, P. Pattanaik and K. Suzumura, eds) Choice, Welfare, and Development: A Festchrift in Honour of Amartya K. Sen, Oxford: Clarendon Press, pp. 169–81.

Bardhan, P. K. 2000. “Irrigation and cooperation: an empirical analysis of 48 irrigation communities in South India”, Economic Development and Cultural Change, vol. 48, pp. 847–65.

Beach, R. H., Poulos, C., and S. K. Pattanayak. 2007. “Agricultural Household Response to Avian Influenza Prevention and Control Policies.” *Journal of Agricultural and Applied Economics* 39: 301.

Bhat, M. G., and R. G. Huffaker. 2007. “Management of a transboundary wildlife population: A self-enforcing cooperative agreement with renegotiation and variable transfer payments.” *Journal of Environmental Economics and Management*, 53(1), 54-67.

Brauer, F., and C. Castillo-Chavez. 2001. “Mathematical Models in Population Biology and Epidemiology.” *Texts in Applied Mathematics* 40, Springer-Verlag, New York.

Brown, C., L. Lynch, and D. Zilberman. 2002. “The Economics of Controlling Insect Transmitted Plant Diseases.” *American Journal of Agricultural Economics*

84 (2):279-291.

Cabaleiro, C., C. Couceiro, S. Pereira, M. Barrasa, and A. Segura. 2008. “Spatial analysis of epidemics of Grapevine leafroll associated virus-3.” *European Journal of Plant Pathology* 121:121-130.

Cabaleiro, C. and A. Segura. 2007. “Some characteristics of the transmission of grapevine leafroll associated virus 3 by Planococcus citri Risso.” *European Journal of Plant Pathology* 103, no. 4: 373-378.

——. 2006. “Temporal analysis of grapevine leafroll associated virus 3 epidemics.” *European Journal of Plant Pathology* 114:441–446

Cane, V., and R. McNamee. 1982. “The Spread of Infection in a Heterogeneous Population.” *Journal of Applied Probability* 19: 173-184.

Ceddia, M. G., Bartlett, M., De Lucia, C., and Perrings, C. 2011. On the regulation of spatial externalities: coexistence between GM and conventional crops in the EU and the ‘newcomer principle’. Australian journal of agricultural and resource economics, 55(1), 126-143.

Chan, M-S, and M. J. Jeger. 1994. “An Analytical Model of Plant Virus Disease Dynamics with Roguing and Replanting.” *Journal of Applied Ecology* 31: 413-27.

Charles, J., K. Froud, R. van den Brink, and D. Allan. 2009. “Mealybugs and the spread of Grapevine leafroll-associated virus 3(GLRaV-3) in a New Zealand Vineyard.” *Australasian Plant Pathology* 6:576-83

Constable, F.E., Connellan, J., Nicholas, P. and B. C. Rodoni. 2012. “Comparison of Enzyme Linked Immunosorbent Assays and Reverse-Transcription Polymerase Chain Reaction for the Reliable Detection of Australian Grapevine Viruses in

Two Climates during Three Growing Seasons." *Australian Journal of Grape and Wine Research* 18: 239–244.

Corp, M. K., and T. Darnell. 2002. "Conflict-Laden Issues: A Learning Opportunity." *Journal of Extension*, 40 (1). Available at http://www.joe.org/joe/2002february/rb1.php. Last accessed 6/25/2014.

Cox, D. R. 1959. "The Analysis of Exponentially Distributed Life-Times with Two Types of Failure." *Journal of the Royal Statistical Society. Series B* 21: 411-421.

Daane, K. M., R. P.P. Almeida, V. A. Bell, J. T.S. Walker, M. Botton, M. Fallahzadeh, M. Mani, J.L. Miano, R. Sforza, V.M. Walton and T. Zaviezo. 2012. "Biology and Management of Mealybugs in Vineyards." In *Arthropod Management in Vineyards*: *Pests, Approaches, and Future Directions* N.J. Bostanian et al. (eds.), pp. 271-307. Springer Netherlands.

Dayton-Johnson, J., and Bardhan, P. 2002. "Inequality and conservation on the local commons: a theoretical exercise." *The Economic Journal*, 112 (481), 577-602.

Demont, M., Dillen, K., Daems, W., Sausse, C., Tollens, E., and Mathijs, E. 2009. "On the proportionality of EU spatial *ex ante* coexistence regulations." Food Policy, 34(6), 508-518.

Diagnostics, A.C. 2012. *ELISA tests for Plant Viruses: Grapevine leafroll-associated virus - 3 (GLRaV-3) - DAS ELISA*. Available at http://www.acdiainc.com/GLRaV-3.htm. Last accessed 10/12/2012.

Epanchin-Niell, R. S. and J.E. Wilen. 2012. "Optimal Spatial Control of Biological Invasions." *Journal of Environmental Economics and Management* 63: 260-270.

Fenichel, E. P. and Horan R. D. 2007. "Gender-Based Harvesting in Wildlife Disease Management." *American Journal of Agricultural Economics* 89:904-920.

Foroutan, Pirouz. 2003. "An Agent-based Modeling of Raccoon Rabies Epidemic and its Economic Consequences," Doctoral Dissertation, Ohio State University, Columbus, Ohio. Available at http://www.carnivoreconservation.org/files/thesis/foroutan_2003_phd.pdf. Last accessed 7/6/2013.

Fuchs, M., P. Marsella-Herrick, G.M. Loeb, T.E. Martinson, and H.C. Hoch. 2009. "Diversity of Ampeloviruses in Mealybug and Soft Scale Vectors and In Grapevine Hosts from Leafroll-Affected Vineyards." *Phytopathology* 99:1177-1184.

Fuchs, M. 2007. "Grape Leafroll Disease." Cornell University and the New York State IPM

Program. Available at http://www.nysipm.cornell.edu/factsheets/grapes/diseases. Last accessed 5/6/2013.

Fuchs, M., P. Marsella-Herrick, G.M. Loeb, T.E. Martinson, and H.C. Hoch. 2009. "Diversity of ampeloviruses in mealybug and soft scale vectors and in grapevine hosts from leafroll-affected vineyards." *Phytopathology* 99:1177-1184.

Fuller, K. B., J. M. Alston, and J. N. Sanchirico. 2011. "Spatial Externalities and Vector-Borne Plant Diseases: Pierce's Disease and the Blue-Green Sharpshooter in the Napa Valley." In *2011 Annual Meeting, July 24-26, 2011, Pittsburgh, Pennsylvania*, no. 103865. Agricultural and Applied Economics Association.

Freeborough M-J and J. Burger. 2006. Leafroll: Economic implications. Wynboer. Accessed 4/2/2011 at http://www.wynboer.co.za/recentarticles/200812-leafroll.php3

Gibson, G. J. 1997. "Markov chain Monte Carlo methods for fitting spatiotemporal stochastic models in plant epidemiology." *Journal of the Royal Statistical Society: Series C (Applied Statistics)*, 46(2), 215-233.

Gilbert, N. and P. Terna. 2000. "How to Build and Use Agent-Based Models in Social Science." *Mind and Society* 1: 57-72.

Golino, D.A., E. Weber, S.T. Sim, and A. Rowhani. 2008. "Leafroll disease is spreading rapidly in a Napa Valley vineyard." *California Agriculture* 62:156-160.

Golino, D., Sim, S., Gill, R., and A. Rowhani, A. 2002. "California mealybugs can spread grapevine leafroll disease." *California Agriculture* 56: 6, 196-201.

Goheen, A.C., and J.A. Cook. 1959. "Leafroll (red-leaf or rougeau) and its effects on vine growth, fruit quality, and yields". *American Journal of Enology and Viticulture* 10:4:173-181

Grasswitz, T. R., and D. G. James (2008). "Movement of grape mealybug, Pseudococcus maritimus, on and between host plants." *Entomologia Experimentalis et Applicata*, 129(3), 268-275.

Habili, N., C. F. Fazeli, A. Ewart, R. Hamilton, R. Cirami, P. Saldarelli, A. Minafra, and M. A. Rezaian. 1995. "Natural spread and molecular analysis of grapevine leafroll-associated virus 3 in Australia." *Phytopathology* 85, no. 11: 1418

Hall, D. C. and R. B. Norgaard. 1973. "On the timing and application of pesticides." *American Journal of Agricultural Economics* 55, 198-201.

Holmes, E. E., M. A. Lewis, J. E. Banks, and R. R. Veit. 1994. "Partial Differential Equations in Ecology: Spatial Interactions and Population Dynamics." *Ecology* 75: 17-29.

Horan, R. D., E. P. Fenichel, C. A. Wolf, and B. M. Gramig. 2010. "Managing Infectious Animal Disease Systems." *Annual Review of Resource Economics* 2:101-124.

Horan, R.D., and C.A. Wolf. 2005. "The Economics of Managing Infectious Wildlife Disease." *American Journal of Agricultural Economics* 87: 537–551.

Johnson, R. 2009. "Specialty Crops: 2008 Farm Bill Issues." Congressional Research Service Report RL33520. Available at: ttp://www.nationalaglawcenter.org/assets/crs/RL33520.pdf. Last accessed 5/6/2013.

Jooste, A. E. C., G. Pietersen and J. T. Burger. 2011. "Distribution of Grapevine Leafroll Associated Virus-3 Variants in South African Vineyards." *European Journal of Plant Pathology* 131:371–381.

Kanbur, R. and K. Michael. "Jeux Sans Frontieres: Tax Competition and Tax Coordination When Countries Differ in Size." American Economic Review, September 1993, 83(4), pp. 877-92.

Kaye-Blake, W., F. Y. Li, A. McLeish Martin, A. McDermott, H. Neil, and S. Rains. 2009. "A Review of Multi-Agent Simulation Models in Agriculture." Paper

presented at the 2009 NZARES Conference Tahuna Conference Centre – Nelson, New Zealand. August 27-28, 2009.

Khwaja, A. I. 2007. “Can good projects succeed in bad communities?” Harvard University, Kennedy School of Government Working Papers.

Klaassen, V. A., S. T. Sim, G. S. Dangl, F. Osman, M. Al Rwahnih, A. Rowhani, and D. A. Golino. 2011. "Vitis californica and Vitis californica× Vitis vinifera Hybrids are Hosts for Grapevine leafroll-associated virus-2 and-3 and Grapevine virus A and B." *Plant Disease* 95, no. 6: 657-665.

Kotz, S. and J. R. van Dorp. 2004. “Beyond Beta Other Continuous Families of Distributions with Bounded Support and Applications.” Singapore: World Scientific 289 pp.

Le Maguet, J., J. J. Fuchs, J. Chadœuf, M. Beuve, E. Herrbach, and O. Lemaire. 2013. “The role of the mealybug Phenacoccus aceris in the spread of Grapevine leafroll-associated virus− 1 (GLRaV-1) in two French vineyards.” *European Journal of Plant Pathology*, 135(2), 415-427.

Luminex 2010. “Overcoming the Cost and Performance Limitations of ELISA with xMAP® Technology. xMAP® Technology.” Technical Note. Luminex Corporation, Austin, Texas.

Lybbert, T. J., Magnan, N., and W. D. Gubler (2010). Powdery Mildew Risk And Forecasting In Wine Grapes: Do Growers Change Risk Management Strategies In Response To Disease Forecasts?. In *2010 Annual Meeting, July 25-27, 2010, Denver, Colorado* (No. 61745). Agricultural and Applied Economics Association.

Maree, H. J., R. P. Almeida, R. Bester, K. M. Chooi, D. Cohen, V. V. Dolja, M. F. Fuchs (2013). "Grapevine leafroll-associated virus 3." *Frontiers in microbiology* 4:82

Marshall, E. P., and F. R. Homans. 2004. "A Spatial Analysis of the Economic and Ecological Efficacy of Land Retirement." *Environmental Modeling and Assessment* 9: 65-75.

Martelli, G.P., and E. Boudon-Padieu. 2006. "Directory of infectious diseases of grapevines. International Centre for Advanced Mediterranean Agronomic Studies." *Options Méditerranéennes* Ser. B, Studies and Research 55:59-75.

Martin, R.R., K.C. East well, A. Wagner, S. Lamprecht, and I.E. Tzanetakis. 2005. "Survey for viruses of grapevine in Oregon and Washington. *Plant Disease*." 89:763-766.

Martinson, T.E., M. Fuchs, G. Loeb, and H.C. Hoch. 2008. "Grapevine leafroll: An increasing problem in the Finger Lakes, the US and the world." *Finger Lakes Vineyard Notes* 6:6-11.

Medlock, J., and A. P. Galvani. 2009. "Optimizing Influenza Vaccine Distribution." *Science* 325: 1705-1708.

Miller, J. H. and S. E. Page, .2007. "Complex Adaptive Systems: An Introduction to Computational Models of Social Life." Princeton University Press, Princeton, USA, 284 pp.

Munro, G. R. 1979. "The optimal management of transboundary renewable resources." *Canadian Journal of Economics*, 355-376.

Muthoo, A. 1999. *Bargaining theory with applications*. Cambridge University Press.

Nash, J. 1953. “Two-person cooperative games.” *Econometrica: Journal of the Econometric Society*, 128-140.

Nimmo-Bell. 2006. “The economic effects and financial impact of GLRaV-3.”. Nimmo-Bell and Company, Wellington, NZ, 18 pp.

Olson, M. 1965.The Logic of Collective Action. Cambridge, MA: Harvard University Press.

Over de Linden AJ, Chamberlain EE. 1970. Effect of grapevine leafroll virus on vine growth and fruit yield and quality. New Zealand Journal of Agricultural Research 13: 689-698.

Parker, D. C., and Munroe, D. K. 2007. “The geography of market failure: edge-effect externalities and the location and production patterns of organic farming.” Ecological Economics, 60(4), 821-833.

Parker, D.C., September 2000. Edge-effect externalities: Theoretical and empirical implications of spatial heterogeneity. Ph.D. thesis, University of California at Davis.

Pietersen, G. 2006. “Spatio-temporal dynamics of grapevine leafroll disease in Western Cape vineyards.” *Extended Abstracts*, 15th Meeting of the International Council for the Study of Virus and Virus-like Diseases of the Grapevine, 2006. Stellenbosch, South Africa, pp 126-127.

Rahmandad, H. and J. Sterman. 2008. “Heterogeneity and Network Structure in the Dynamics of Diffusion: Comparing Agent-Based and Differential Equation Models.” *Management Science* 54: 998–1014.

Rayapati, A. N., S. O'Neil and D. Walsh. 2008. "Grapevine leafroll disease." *WSU Extension Bulletin EB2027E*.

Regev, U., Gutierrez, A. P., and G. Feder. 1976. "Pests as a common property resource: A case study of alfalfa weevil control." *American Journal of Agricultural Economics*, 58(2), 186-197.

Rich, K. M., and A. Winter-Nelson. 2007. "An Integrated Epidemiological-Economic Analysis of Foot and Mouth Disease: Applications to the Southern Cone of South America." *American Journal of Agricultural Economics* 89: 682-697.

Rich, K. M., A. Winter-Nelson, and N. Brozović, N. 2005. "Modeling Regional Externalities with Heterogeneous Incentives and Fixed Boundaries: Applications to Foot and Mouth Disease Control in South America." *Applied Economic Perspectives and Policy* 27: 456-464.

Roth, A., Y. Pan, Z. Yu, and R. Doluschitz. 2009. "Using Multi-Agent Modeling Technique to Regionalize Key Processes and Patterns of Sustainable Agricultural Cropping Systems in the North China Plain." In *2009 Conference, August 16-22, 2009, Beijing, China*, no. 51747. International Association of Agricultural Economists.

Rubenstein, A. 1982." Perfect equilibrium in a bargaining model". *Econometrica: Journal of the Econometric Society*, 97-109.

Sanchirico, J. and J. Wilen. 1999. "Bioeconomics of Spatial Exploitation in a Patchy Environment." *Journal of Environmental Economics and Management* 37: 129-150.

——. 2005. “Optimal Spatial Management of Renewable Resources: Matching Policy Scope to Ecosystem Scale.” *Journal of Environmental Economics and Management* 50: 23–46.

Saphores, J.D.M. 2000. “The Economic Threshold with a Stochastic Pest Population: a Real Options Approach.” *American Journal of Agricultural Economics* 82: 541–555.

Sharov, A. A., and Liebhold, A. M. 1998. “Bioeconomics of Managing the Spread of Exotic Pest Species with Barrier Zones.” *Ecological applications* 8: 833-845.

Skinkis, P., A. J. Dreves, V. M. Walton, and R. R. Martin. 2009. “Field monitoring for grapevine leafroll virus and mealybug in Pacific Northwest vineyards.” Oregon State University, Extension Service. EM 8985.

Smith, M. D., J. N. Sanchirico, and J. E. Wilen. 2009. “The Economics of Spatial-Dynamic Processes: Applications to Renewable Resources.” *Journal of Environmental Economics and Management* 57: 104-121.

Sumaila, U. R. 1997. “Cooperative and non-cooperative exploitation of the Arcto-Norwegian cod stock.” *Environmental and Resource Economics*, 10(2), 147-165.

Sun, G-Q, Q-X Liu, Z. Jin, A. Chakraborty, and B-L. Li. 2010. “Influence of Infection Rate and Migration on Extinction of Disease in Spatial Epidemics.” *Journal of Theoretical Biology* 264: 95-103.

Tahvonen, O. 2009. “Economics of Harvesting Age-Structured Fish Populations.” *Journal of Environmental Economics and Management* 58: 281-299.

Teose, M., K. Ahmadizadeh, E. O'Mahony, R. L. Smith, Z. Lu, S. E. Ellner, C. Gomes, Y. Grohn 2011. “Embedding System Dynamics in Agent Based Models for Complex Adaptive Systems”. Twenty-Second International Joint Conferences on Artificial Intelligence, Barcelona, Spain, July 16-22, 2011.

Tesfatsion, L., and K. L. Judd (ed.). 2006. Handbook of Computational Economics, Elsevier, edition 1, volume 2, number 2.

Tsai, C.W., A. Rowhani, D.A. Golino, K.M. Daane, and R.P.P. Almeida. 2010. “Mealybug Transmission of Grapevine Leafroll Viruses: an Analysis of Virus-Vector Specificity.” *Phytopathology* 100:830-834.

Tsai, C-W., J. Chau, L. Fernandez, D. Bosco, K. M. Daane, and R. P. P. Almeida. 2008. “Transmission Of Grapevine Leafroll-Associated Virus 3 by the Vine Mealybug (Planococcus ficus).” *Phytopathology* 98: 1093-1098.

Walker, J.T.S. Charles, J.G. Froud, K.J., Connolly, P. 2004. Leafroll virus in vineyards: modeling the spread and economic impact. Report to New Zealand Winegrowers Limited: 19pp

Walton, V. M, A.J. Dreves, P. Skinkis, C. Kaiser, M. Buchanan, R. Hilton, B.R. Martin, S. Castagnoli and S. Renquist. 2009. “Grapevine Leafroll Virus and Mealybug Prevention
and Management in Oregon Vineyards.” EM 8985, Oregon State University Extension Service.

Walton, V. M., and K. L. Pringle. 1999. “Effects of Pesticides Used on Table Grapes on the Mealybug Parasitoid *Coccidoxenoides peregrinus* (Timberlake)

(Hymenoptera: Encyrtidae).” *South African Journal for Enology and Viticulture* 20: 31-34.

Warr, P. G. (1983). The private provision of a public good is independent of the distribution of income. *Economics letters*, *13*(2), 207-211.

White, G. 2008. “Cost of establishment and production of vinifera grapes in the Finger Lakes region of New York.” College of Agriculture and Life Sciences, Cornell University, Ithaca, NY.

Wilen, J. 2007. “Economics of Spatial–Dynamic Processes.” *American Journal of Agricultural Economics* 89: 1134–1144.

Wolf, T. K. 2008. “Wine Grape Production Guide for Eastern North America.” Natural Resource, Agriculture, and Engineering Service (NRAES) Cooperative Extension, Ithaca, NY.

Wolfram, S. (ed.). 1986. “Theory and Applications of Cellular Automata.” World Scientific, Singapore.

Yassemi, S., S. Dragićević, and M. Schmidt. 2008. “Design and Implementation of an Integrated GIS-Based Cellular Automata Model to Characterize Forest Fire Behaviour.” *Ecological Modelling* 210: 71-84.